2004 - Present	**Clinical Assistant Professor** **Department of Pathology, Wayne State University School of Medicine** Detroit, Michigan
2005 - Present	**Clinical Assistant Professor** **Department of Fundamental and Applied Sciences, Wayne State University** Detroit, Michigan
2006 - Present	**Staff Physician, Department of Pathology** **Port Huron Hospital** Port Huron, Michigan
2008 - Present	**Adjunct Faculty / Instructor** Macomb Community College Warren, Michigan
2009 - Present	**Clinical Educator** Michigan State University School of Medicine East Lansing, Michigan

CONSULTING POSITIONS

2006 - Present	**Medicolegal and Forensic Science Consultant** **MSNBC, CNN, CNN Headline News, Court TV, Fox News Channel** New York, New York and Altanta, Georgia
2009 - Present	**Forensic Pathology and Forensic Science Consultant** **CSI (Crime Scene Investigation) and CSI Miami, CBS Television Network** Los Angeles, California

PREVIOUS MEDICAL EXAMINER APPOINTMENTS

2001 - 2004	**Associate Medical Examiner, Medical Examiner Department** Hillsborough County, Florida
2001 - 2004	**Associate Director, Forensic Pathology Fellowship Training Program** Hillsborough County, Florida
2003 - 2004	**Associate Medical Examiner, District 12 Medical Examiner Department**

	Sarasota, Manatee and DeSoto Counties, Florida
2000 - 2001	**Associate Medical Examiner, Medical Examiner Department** Miami-Dade County, Florida

OTHER PREVIOUS APPOINTMENTS

2010 - 2013	**Clinical Laboratory Director, Health Department** St. Clair County, Michigan
2004 - 2005	**Forensic Science Instructor, Criminal Justice Department** St. Petersburg College, St. Petersburg, Florida
2002 - 2005	**Consultant Forensic Pathologist, Tampa Pathology Laboratories** Tampa, Florida
2001 - 2004	**Assistant Professor of Pathology and Laboratory Medicine** **University of South Florida, College of Medicine** Tampa, Florida
1999 - 2000	**Chief Resident, Department of Pathology** **Rush Presbyterian-St. Luke's Medical Center** Chicago, Illinois
1998 - 2000	**Staff Pathologist, Christ Hospital and Medical Center** Oak Lawn, Illinois
1998 - 2000	**Research Associate, Rush Alzheimer's Disease Center** Chicago, Illinois
1997 - 2000	**Pathology Laboratory Instructor, Rush Medical College** Chicago, Illinois
August 1987	**Recovery Team, Northwest Airlines Plane Crash - Flight 255** Detroit, Michigan

MEDICAL LICENSES

- Michigan: 4301081020 - 2002 to present
- Florida: ME 82382 - 2001 to present
- Illinois: 36-096963 - 1996 - 2003 (Inactive Status)

EDITORIAL BOARD of EDITORS

2005 - Present	**American Journal of Forensic Medicine and Pathology** Philadelphia, Pennsylvania

COMMITTEE APPOINTMENTS

2006 - Present	**Anatomic Pathology Assistant Program, Advisory Committee** Wayne State University, Detroit, Michigan
2005 - Present	**Michigan Child Death Review, Consultant** St. Clair County, Michigan
2004 - Present	**Michigan Child Death Review, Consultant** Macomb County, Michigan
2002 - 2004	**Pathology Residency Committee, University of South Florida College of Medicine** Tampa, Florida
2002 - 2004	**Florida Child Protection Team, Consultant** Hillsborough County, Florida
2002 - 2004	**Disciplinary Hearing Officer** Hillsborough County, Florida
1997 - 2000	**Admissions Committee, Rush Medical College** Chicago, Illinois
1997 - 2000	**Surgical Procedure Review Committee, Rush Presbyterian - St. Luke's Medical Center** Chicago, Illinois

AWARDS / ACHIEVEMENTS

May, 2009	**Distinguished Service Award, Port Huron Police Department**
January, 2009	**Featured Medical Examiner – *Dead Men Talking*** Three Part Documentary Series - MSNBC Cable Network

INVITED LECTURES / PANEL DISCUSSIONS / SEMINARS

- *Forensic Investigation of Fire Deaths:* St. Clair County Fire Training Seminar, Port Huron, Michigan, January 17, 2015
- *Forensic Death Investigation:* Wayne State University Dept. of Fundamental and Applied Sciences, Detroit, Michigan, July 2, 2014
- *Forensic Pathology and Scene Investigation:* Detective Training Seminar, Mt. Clemens, Michigan, March 26, 2014
- *Child Deaths; Investigation Approach and Pitfalls:* Macomb County Sheriff Department, Mt. Clemens, Michigan, October 25, 2013
- *Wound Pattern Recognition and Injury Analysis:* Wayne State University Dept. of Fundamental and Applied Sciences, Detroit, Michigan, July 14, 2013
- *Forensic Pathology – Application for First Responders*, Training Series, St. Clair Shores Fire Department, St. Clair Shores, Michigan, May 28, 2013
- *Infant and Child Death Investigation:* CTC Training Specialties, Clinton Township, Michigan, October 9, 2012
- *Forensic Death Investigation:* Wayne State University Dept. of Fundamental and Applied Sciences, Detroit, Michigan, July 18, 2012
- *Asphyxiation, Rape, Homicide:* 35th Annual Medicolegal Investigation of Death Seminar, Detroit, Michigan, May 4, 2012
- *Forensic Case Presentation:* 35th Annual Medicolegal Investigation of Death Seminar, Detroit, Michigan, May 3, 2012
- *Death Investigation in Michigan-What All Residents Should Know:* Grosse Pointe Crisis Club, February 16, 2012
- *Forensic Pathology and Duties of Medical Examiner:* Wake-Up Wednesday, Grosse Pointe Yacht Club, Grosse Pointe, Michigan, December 14, 2011
- *Death Investigation, Injury Analysis and Evidence Collection for First Responders:* Southwest Michigan 3rd Annual Botsford Hospital EMS Expo, Novi, Michigan, October 12, 2011

- *Asphyxiation, Rape, Homicide:* 35th Annual Medicolegal Investigation of Death Seminar, Detroit, Michigan, May 6, 2011

- *Forensic Case Presentation:* 35th Annual Medicolegal Investigation of Death Seminar, Detroit, Michigan, May 5, 2011

- *Wound Pattern Recognition and Injury Analysis:* Wayne State University Dept. of Fundamental and Applied Sciences, Detroit, Michigan, July 14, 2010

- *Medical Examiner Scene Investigator (MESI) Training Program, St. Clair County:* Course Director, Port Huron Hospital, Port Huron, Michigan, May, 2010

- *Interpretation of Patterned Injuries:* Sexual Assault Nurse Examiner (SANE) Seminar, Mt. Clemens, Michigan, April 28, 2010

- *Rape/Homicide and Collection of Evidence:* 34th Annual Medicolegal Investigation of Death Seminar, Detroit, Michigan, April 21, 2010

- *Interpretation of Injuries Caused by Firearms,* 34th Annual Medicolegal Investigation of Death Seminar, Detroit, Michigan, April 22, 2010

- *Forensic Pathology and Death Investigation,* Science Seminar, Grosse Pointe North High School, Grosse Pointe, Michigan, December 17, 2009

- *Forensic Pathology, Toxicology and Death Investigation:* Year II Pathology Course, Wayne State University School of Medicine, Detroit, Michigan, November 19, 2009

- *Forensic Pathology and Death Investigation,* Science Seminar, Grosse Pointe North High School, Grosse Pointe, Michigan, May 20, 2009

- *Forensic Pathology and Medical Examiners: What the Public Needs to Know,* 4th Annual Crime Victims' Rights Week Seminar, Clinton Township, Michigan, April 30, 2009

- *Interpretation of Injuries Caused by Firearms,* 33rd Annual Medicolegal Investigation of Death Seminar, Detroit, Michigan, April 24, 2009

- *Rape / Homicide and Collection of Evidence:* 33rd Annual Medicolegal Investigation of Death Seminar, Detroit, Michigan, April 24, 2009

- *Investigation of Deaths in Childhood - From SIDS to Shaking*: 33rd Annual Medicolegal Investigation of Death Seminar, Detroit, Michigan, April 23, 2009

- *Interpretation of Gunshot Wound and Sharp Force Injuries:* Department of Pathology, Wayne State University School of Medicine, Detroit, Michigan, April 7, 2008

- *Introduction to Forensic Pathology-Wound Pattern Recognition:* Basic Detective School, Oakland Police Academy, Auburn Hills, Michigan, March 27, 2009

- *Interpretation of Patterned Injuries:* Sexual Assault Nurse Examiner (SANE) Seminar, Mt. Clemens, Michigan, January 13, 2009

- *Rape-Homicide and Collection / Interpretation of Evidence:* 3rd Annual Medicolegal Investigation of Death Seminar, Las Vegas, Nevada, January 7, 2009

- *Interpretation of Injuries Caused by Firearms,* 3rd Annual Medicolegal Investigation of Death Seminar, Las Vegas, Nevada, January 7, 2009

- *Investigating Deaths in Childhood – From SIDS to Shaking:* 3rd Annual Medicolegal Investigation of Death Seminar, Las Vegas, Nevada, January 6, 2009

- *Forensic Pathology, Toxicology and Death Investigation:* Year II Pathology Course, Wayne State University School of Medicine, Detroit, Michigan, November 21, 2008

- *Insights into the Diagnosis of Cause of Death:* Innovations in Emergency Medicine (Beaumont Hospital), Beverly Hills, Michigan, October 24, 2008

- *Forensic Death Investigation:* Wayne State University Dept. of Fundamental and Applied Sciences, Detroit, Michigan, July 9, 2008

- *Investigation of Deaths in Childhood - From SIDS to Shaking*: 32nd Annual Medicolegal Investigation of Death Seminar, Detroit, Michigan, April 23, 2008

- *Rape/Homicide and Collection of Evidence:* 32nd Annual Medicolegal Investigation of Death Seminar, Detroit, Michigan, April 25, 2008

- *Interpretation of Gunshot Wound and Sharp Force Injuries:* Department of Pathology, Wayne State University School of Medicine, Detroit, Michigan, April 7, 2008

- *Investigation Drowning Deaths:* Continuing Education, Sterling Heights Fire Department, Sterling Heights, Michigan, March 11, 2008

- *A Day in the Life of a Forensic Pathologist:* American Academy of Forensic Sciences, Student Academy, 60th Anniversary Meeting, Washington DC, February 19, 2008

- *Recognizing Child Abuse: What Schools and Educators Need to Know:* Child Protection Law Workshop (Panel Discussion), Macomb Intermediate School District, Clinton Township, Michigan, February 8, 2008

- *Rape-Homicide and Collection/Interpretation of Evidence:* 2nd Annual Medicolegal Investigation of Death Seminar, Las Vegas, Nevada, December 7, 2007

- *Case Review in Drowning Deaths and Bodies Recovered from Water:* 2nd Annual Medicolegal Investigation of Death Seminar, Las Vegas, Nevada, December 6, 2007

- *Investigation of Deaths in Childhood: From SIDS to Shaking:* Pediatric Pathology Department, Children's Hospital of Michigan, Detroit, Michigan, November 30, 2007

- *Forensic Pathology, Toxicology and Death Investigation:* Year II Pathology Course, Wayne State University School of Medicine, Detroit, Michigan, November 16, 2007

- *Forensic Pathology for the Boards*: Osler Institute, Pathology Board Examination Review Course, Tampa, Florida, September 30, 2007

- *Wound Pattern Recognition: An Introduction to Forensic Pathology and Injury Analysis:* Wayne State University Dept. of Fundamental and Applied Sciences, Detroit, Michigan, July 18, 2007

- *Investigation and Analysis of Firearm Wounds:* 31st Annual Medicolegal Investigation of Death Seminar, Detroit, Michigan, April 27, 2007

- *Introduction to Forensic Pathology*: Department of Pathology, Wayne State University, Detroit, Michigan, January 26, 2007

- *Investigation of Deaths in Childhood: From SIDS to Shaking (Part 2):* Pediatric Pathology Department, Children's Hospital of Michigan, Detroit, Michigan, December 15, 2006

- *Analysis of Gunshot Wounds – Wound Pattern Recognition:* 1st Annual Medicolegal Investigation of Death Seminar, Las Vegas, Nevada, November 30, 2006

- *Investigation of Drowning Deaths and Bodies Recovered from Water:* 1st Annual Medicolegal Investigation of Death Seminar, Las Vegas, Nevada, November 30, 2006

- *Investigation of Deaths in Childhood: From SIDS to Shaking (Part 1):* Pediatric Pathology Department, Children's Hospital of Michigan, Detroit, Michigan, November 17, 2006

- *Injury Analysis and Wound Pattern Recognition:* Forensic Nurse Examiners Conference, Warren, Michigan, November 2, 2006

- *Forensic Pathology for the Boards*: Osler Institute, Pathology Board Examination Review Course, Tampa, Florida, October 1, 2006

- *Wound Pattern Recognition: An Introduction to Forensic Pathology and Injury Analysis:* Wayne State University, Department of Fundamental and Applied Sciences, Detroit, Michigan, June 27, 2006

- *Forensic Pathology for the Boards*: Osler Institute, Pathology Board Examination Review Course, Tampa, Florida, May 31, 2006

- *Analysis of Gunshot Wounds – Wound Pattern Recognition:* 30th Annual Medicolegal Investigation of Death Seminar, Detroit, Michigan, April 28, 2006

- *Wound Pattern Recognition and Analysis:* National Legal Aid and Defender Association: Life in the Balance, Philadelphia, Pennsylvania, March 5, 2006

- *Forensic Case Consultation – Panel Discussion:* National Legal Aid and Defender Association: Life in the Balance, Philadelphia, Pennsylvania, March 5, 2006

- *Evaluating Gunshot Wounds & Sharp Force Injuries*: National Legal Aid and Defender Association: Life in the Balance, Philadelphia, Pennsylvania, March 6, 2006

- *Wound Identification and Pattered Injuries*: Sexual Assault Nurse Examiner Conference, Macomb County Community College, January 18, 2006

- *Gunshot Wounds: What Do They Tell Us?* Georgia Public Defender Standards Council, Forensic Science Seminar, Atlanta, Georgia, November 9, 2005

- *Drowning and Bodies Recovered from Water; Solving the Puzzle:* Michigan Association of Medical Examiners, Annual Meeting, Mt. Pleasant, Michigan, October 30, 2005

- *Death Investigation and Wound Pattern Analysis:* Michigan Police Academy Detective Training, Oakland County Community College, October 27, 2005

- *Postmortem Changes: Recognizing Injury v. Artifact:* 12th Annual Medicolegal Death Investigation Conference, Collinsville, Illinois, August 17, 2005

- *Investigation of Drowning Deaths and Bodies Recovered from Water:* 12th Annual Medicolegal Death Investigation Conference, Collinsville, Illinois, August 17, 2005

- *Interpretation of Gunshot Wounds and Sharp Force Injuries. 12th* Annual Medicolegal Death Investigation Conference, Collinsville, Illinois, August 17, 2005

- *Wound Pattern Recognition: An Introduction to Injury Analysis:* Wayne State University Dept. of Fundamental and Applied Sciences, Detroit, Michigan, June 28, 2005

- *Child Abuse: Interpretation of Injuries and Distinction from Natural and Accidental Deaths:* Pediatric Sexual Assault Nurse Examiner Training Seminar, Fraser, Michigan, May 19, 2005

- *Analysis of Gunshot Wounds – Wound Pattern Recognition:* 29th Annual Medicolegal Investigation of Death Seminar, Detroit, Michigan, April 21, 2005

- *Investigation of Drowning Deaths and Bodies Recovered from Water:* 29th Annual Medicolegal Investigation of Death Seminar, Detroit, Michigan, April 20, 2005

- *Interpretation of Forensic Evidence:* National Legal Aid and Defender Association: Life in the Balance, New Orleans, Louisiana, March 20, 2005

- *Evaluating Gunshot Wounds & Sharp Force Injuries*: National Legal Aid and Defender Association: Life in the Balance, New Orleans, Louisiana, March 20, 2005

- *Forensic Case Consultation:* National Legal Aid and Defender Association: Life in the Balance, New Orleans, Louisiana, March 20, 2005

- *Forensic Case Analysis:* St. Clair County, Medical Examiner Scene Investigator Conference, Marysville, Michigan, February 22, 2005

- *Injury Analysis and Wound Pattern Recognition:* Forensic Nurse Examiners Conference, Warren, Michigan, January 17, 2005

- *Forensic Pathology for the Boards*: Osler Institute, Pathology Board Examination Review Course, Tampa, Florida, October 10, 2004

- *Interpretation of Gunshot and Shotgun Wounds:* St. Clair County Forensic Training Seminar, Marysville, Michigan, October 7, 2004

- *Thermal Injuries and Bodies Recovered From Water:* St. Clair County Forensic Training Seminar, Marysville, Michigan, October 7, 2004

- *Investigation of Deaths in Childhood:* St. Clair County Forensic Training Seminar, Marysville, Michigan, October 6, 2004

- *Forensic Pathology and Interpretation of Wound Patterns:* 11th Annual International Homicide Investigators Association, Clearwater, Florida, August 2, 2004

- *Forensic Pathology for the Boards*: Osler Institute, Pathology Board Examination Review Course, Tampa, Florida, June 6, 2004

- *Investigation of Drowning Deaths and Bodies Recovered from Water:* 28th Annual Medicolegal Investigation of Death Seminar, Detroit, Michigan, April 23, 2004

- *Drowning and Bodies In Water*: University of South Florida, Department of Pathology, Tampa, Florida, April 15, 2004

- *Interpretation of Blunt Trauma and Sharp Force Injuries*: 18th Annual Medicolegal Investigation of Death Seminar, Morgantown, West Virginia, March 27, 2004

- *Interpretation of Injuries due to Firearms*: 18th Annual Medicolegal Investigation of Death Seminar, Morgantown, West Virginia, March 27, 2004

- *Understanding the State's Pathology Witness*: National Legal Aid and Defender Association: Life in the Balance, Memphis, Tennessee, March 15, 2004

- *Evaluating Gunshot Wounds & Sharp Force Injuries*: National Legal Aid and Defender Association: Life in the Balance, Memphis, Tennessee, March 14, 2004

- *Forensic Evidence Collection and Interpretation: The Role of the Medical Examiner:* Tampa Bay Area Chiefs of Police, Annual Forensic Science Seminar, Tampa, Florida, March 5, 2004

- *Injury Pattern Recognition and Interpretation:* Death is Different, Florida Association of Criminal Defense Attorneys, 10th Annual Death Penalty Seminar, Palm Beach, Florida, February 21, 2004

- *Medicolegal Aspects of Death Investigation:* St. Petersburg College, Southeastern Public Safety Institute, St. Petersburg, Florida, October 22, 2003

- *Forensic Pathology and Death Investigation:* University of South Florida, Medical Student Colloquium, Tampa, Florida, October 16, 2003

- *Interpretation of Gunshot Wounds*: University of South Florida, Department of Pathology, Tampa, Florida, September 18, 2003

- *Forensic Pathology for the Boards*: Osler Institute, Pathology Board Examination Review Course, Tampa, Florida, May 25, 2003

- *Investigation of Drowning Deaths and Bodies Recovered from Water:* 27th Annual Medicolegal Investigation of Death Seminar, Detroit, Michigan, April 4, 2003

- *Interpretation of the Autopsy Report - Understanding the State's Pathology Witness*: National Legal Aid and Defender Association: Life in the Balance, Austin, Texas, March 18, 2003

- *Evaluating Gunshot Wounds and Sharp Force Injuries*: National Legal Aid and Defender Association: Life in the Balance, Austin, Texas, March 18, 2003
- *Drowning and Bodies In Water*: University of South Florida, Department of Pathology, Tampa, Florida, March 13, 2003

- *Gunshot Wound Evaluation*: University of South Florida, Department of Pathology, Tampa, Florida, October 10, 2002

- *Patterns of Injury in Trauma*: 6th Annual Trauma Symposium, Tampa, Florida, May 17, 2002

- *Introduction to Forensic Pathology*: National Defender Investigator Association, Portland, Oregon, April 18, 2002

- *Investigation of Drowning Deaths and Bodies Recovered from Water*: 26th Annual Medicolegal Investigation of Death Seminar, Detroit, Michigan, April 8, 20

PEER REVIEWED SCIENTIFIC PUBICATIONS

BOOKS

- Spitz WU, **Spitz DJ**. *Medicolegal Investigation of Death*, 4th Edition. Charles C. Thomas, Springfield, Illinois, 2006.

- Gattuso P. Reddy V, David O, **Spitz DJ**, Haber MH. *Differential Diagnosis in Surgical Pathology*, 2nd Edition. W.B. Saunders Company, New York, New York, 2010.

- Haber MH, Gattuso P, **Spitz DJ**, David O. *Differential Diagnosis in Surgical Pathology*. W.B. Saunders Company, New York, New York, 2002.

BOOK CHAPTERS

- Diegel R, Henry T, **Spitz DJ**. Wound Identification and Documentation *in Atlas of Sexual Violence*. Elsevier, St. Louis, Missouri, 2013.

- **Spitz DJ**. Investigation of Bodies in Water *in Spitz and Fisher's Medicolegal Investigation of Death*, 4th Edition. Charles C. Thomas, Springfield, Illinois, 2006.

- Platt MS, **Spitz DJ**, Spitz WU. Investigation of Deaths in Childhood: The Abused Child and Adolescent *in Spitz and Fisher's Medicolegal Investigation of Death*, 4th Edition. Charles C. Thomas, Springfield, Illinois, 2006.
- Spitz WU, **Spitz DJ**. Investigation of Deaths in Childhood: Feticide and Neonaticide *in Spitz and Fisher's Medicolegal Investigation of Death*, 4th Edition. Charles C. Thomas, Springfield, Illinois, 2006.

- **Spitz DJ**. History and Development of Forensic Medicine and Pathology *in Spitz and Fisher's Medicolegal Investigation of Death*, 4th Edition. Charles C. Thomas, Springfield, Illinois, 2006.

- **Spitz DJ**. Identification of Human Remains *in Spitz and Fisher's Medicolegal Investigation of Death*, 4th Edition. Charles C. Thomas, Springfield, Illinois, 2006.

- **Spitz DJ**, Yocom J, Reddy VB. Heart, Pericardium and Blood Vessels *in Differential Diagnosis in Surgical Pathology*. W.B. Saunders Company, New York, New York, 2002.

- **Spitz DJ**, Gattuso P. Ureter, Urinary Bladder and Kidney *in Differential Diagnosis in Surgical Pathology*. W.B. Saunders Company, New York, New York, 2002.

- **Spitz DJ**, Cochran EJ. Central Nervous System in *Differential Diagnosis in Surgical Pathology*. W.B. Saunders Company, New York, New York, 2002.

- Pins MR, Betlej TM, Dysico G, **Spitz DJ**. Male Genitourinary System *in Differential Diagnosis in Surgical Pathology*. W.B. Saunders Company, New York, New York, 2002.

- **Spitz DJ**, Gattuso P. Breast *in Differential Diagnosis in Surgical Pathology*. W.B. Saunders Company, New York, New York, 2002.

- Kapur S, **Spitz DJ**, Reddy VB. Soft Tissue *in Differential Diagnosis in Surgical Pathology*. W.B. Saunders Company, New York, New York, 2002.

- **Spitz DJ**, Gattuso P. Adrenal Gland *in Differential Diagnosis in Surgical Pathology*. W.B. Saunders Company, New York, New York, 2002.

ORIGINAL ARTICLES

- Mateju E, Duchanova S, Kovac P, Moravansky N, **Spitz DJ**. Fatal Case of Rapunzel Syndrome in Neglected Child. *Forensic Science International*, 190:5-7, July 2009.

- Kovac P, Moravansky N, **Spitz DJ**. Child Abuse and Neglect, Minimum for Primary Care Pediatrician. *Practical Pediatrics*, 1:49-51, January 2009.

- Duer WC, **Spitz DJ**, McFarland, S. Relationships between Concentrations of Cocaine Hydrolysates in Peripheral Blood, Heart Blood, Vitreous Humor and Urine. *Journal of Forensic Sciences*, (51)2:421-425, March 2006.

- **Spitz DJ**, Prator PC, Stratton JE, Labiste L, Augenstein JS, Mackinnon J, Phillips J, Singer M, Perdeck E. Neck and Cervical Spine Injuries Caused by Automatic Two-Point Shoulder Restraints: An Analysis of 4 Cases. *Journal of Forensic Sciences*, 50(1):159-163, January 2005.

- **Spitz DJ**, Ouban A. Meningitis Following Gunshot Wound of the Neck. *Journal of Forensic Sciences*, 48(6):1369-1370, November 2003.

- **Spitz DJ**. An Unusual Death in an Asthmatic. *The American Journal of Forensic Medicine and Pathology,* 24(3):271-2, September 2003.

- **Spitz DJ**, Adams VI. Medical Investigation of Pedestrian Deaths. *American Society of Clinical Pathologists, Forensic Pathology Check Sample*, Vol. 44, No. 2, ISSN-1056-5922, May 2002.

- **Spitz DJ**. Drowning and Near-Drowning: Pathophysiology Associated with Freshwater and Saltwater Environments. *American Society of Clinical Pathologists, Forensic Pathology Check Sample,* Vol. 43, No. 3, ISSN-1056-5922, October 2001.

- Hertz G, Reddy VB, Green L, **Spitz DJ**, Massarani-Wafai R, Selvaggi SM, Kluskens L, Gattuso P. Fine Needle Aspiration of Liver Masses: A Multicenter Study of 602 Radiologically Guided FNA. *Diagnostic Cytopathology*, 23(5):326-328, November 2000.

- Orucevic A, Reddy VB, Bakhos R, Selvaggi SM, Green L, **Spitz DJ**, Bitterman P, Gattuso P. Fine Needle Aspiration of Extranodal and Extramedullary Hematopoietic Malignancies. *Diagnostic Cytopathology*, 23(5):318-321, November 2000.

- Betlej TM, **Spitz DJ**, DeCresce RP, Webster RA, Geller J, Maturen A. Performance of the Nichols QuiCk Intraoperative Intact Parathyroid Hormone Assay in Clinical Practice. *Clinical Chemistry*, 46(6):469, July 2000.

- **Spitz DJ**, Reddy VB, Selvaggi SM, Kluskens L, Green L, Gattuso P. Fine Needle Aspiration of Scalp Lesions. *Diagnostic Cytopathology*, 23(1):35-38, July 2000.

- **Spitz DJ**, Reddy VB, Kluskens L, Cohen J, Gattuso P. Fine Needle Aspiration of Intra and Extraocular Masses. *Diagnostic Cytopathology*, 22(3):199-200, March 2000.

- Dowlat K, Fan M, Bloom KJ, **Spitz DJ**, Patel S, Snider H. Occult Metastasis in the Sentinel Lymph Nodes of Patients with Early Breast Carcinoma. *Cancer*, 86(6):990-996, September 1999.

- Gattuso P, Ramzy I, Truong LD, Lanksford KL, Green L, Kluskens L, **Spitz DJ**, Reddy VB. Utilization of Fine Needle Aspiration in the Diagnosis of Metastatic Tumors to the Kidney. *Diagnostic Cytopathology,* 21(1):35-38, July 1999.

- **Spitz DJ**, Maturen A, Cho BL, Webster RA, Betlej TM, DeCresce RP. Performance of the Abbott AxSYM Troponin I Assay and Comparison with CKMB in Clinical Practice. *Clinical Chemistry,* 45(6):501, June 1999.

- **Spitz DJ**, Reddy VB, Gattuso P. Fine Needle Aspiration of Pseudoangiomatous Stromal Hyperplasia of the Breast. *Diagnostic Cytopathology*, 20(5):323-324, May 1999.

- **Spitz DJ**, Reddy VB, Gattuso P. Congenital (Infantile) Myofibromatosis and the Approach to the Differential Diagnosis of Soft Tissue Tumors in Children. *American Society of Clinical Pathologists, Surgical Pathology Check Sample,* Vol. 27, No. 3, ISSN-1091 8655, May 1999.

- **Spitz DJ**. Unrecognized Fatal Liver Injury Caused by a Bicycle Handlebar. *American Journal of Emergency Medicine*, 17:3, 244, May 1999.

- **Spitz DJ**, Spitz WU. Killer Pop Machines. *Journal of Forensic Sciences*, 35(2):490-492, February 1990.

ABSTRACTS

- Moravansky N, **Spitz DJ**, Kovac P, Garala P, Holla B. Child Abuse and Neglect Syndrome: Aspects of Forensic Investigation. *8th Annual Symposium on Forensic Sciences*, Samorin-Cilistov, Slovak Republic, September, 2007.

- Gavala P, Mlynar J. Novomesky F, Morovansky N, **Spitz DJ**, Kovac P, Juricek L. Digital Atlas of Forensic Medicine and Science. *47th Annual Conference of Forensic Medicine.* Modra-Harmonia, Slovak Republic, May 2007.

- Moravansky N, **Spitz DJ**, Kovac P, Garala P, Holla B. Child Abuse and Neglect Syndrome: Aspects of Forensic Investigation. *47th Annual Conference of Forensic Medicine.* Modra-Harmonia, Slovak Republic, May 2007.

- Bloom, KJ, Anderson J, Assad L, **Spitz DJ**, Fan M, Dowlat K. Complete Evaluation of Sentinel Lymph Nodes at 0.25 mm Intervals Utilizing H&E and Cytokeratin Immunohistochemistry. *United States and Canadian Academy of Pathology*, New Orleans, Louisiana, March 2001.

- Dowlat K, Witt TR, Bloom KJ, Fan M, **Spitz DJ**, Oleske D. Detection of Occult Micrometastases by 0.25 mm Sectioning and Cytokeratin Staining of Sentinel Nodes in Early Breast Cancer. *36th Annual American Society of Clinical Oncology*, New Orleans, Louisiana, May 2000.

- Orucevic A, Reddy VB, Bakhos R, Selvaggi SM, Green L, **Spitz DJ**, Bitterman P, Gattuso P. Fine Needle Aspiration of Extranodal and Extramedullary Hematopoietic Malignancies. *47th Annual Scientific Meeting of the American Society of Cytopathology,* Sacramento, California, November 1999.

- **Spitz DJ**, Reddy VB, Bahkos R, Kluskens L, Green L, Selvaggi SM, Gattuso P. Fine Needle Aspiration of Scalp Lesions. *47th Annual Scientific Meeting of the American Society of Cytopathology*, Sacramento, California, November 1999.

- Dowlat K, Fan M, Bloom KJ, **Spitz DJ**. Sentinel Node Biopsy is Superior to Axillary Node Dissection in Staging of Early Breast Cancer. *119th Annual American Surgical Association*, San Diego, California, April 1999.

- Dowlat K, Bloom KJ, Fan M, **Spitz DJ**. Sentinel Node Micrometastases in Early Breast Cancer. *21st Annual San Antonio Breast Cancer Symposium*, San Antonio, Texas, December 1998.

- **Spitz DJ**, Reddy VB, Kluskens L, Cohen J, Gattuso, P. Fine Needle Aspiration of Intra and Extraocular Masses. *American Society of Cytopathology*, Nashville, Tennessee, November 1998.

PROFESSIONAL MEMBERSHIPS

- American Academy of Forensic Sciences, 2001 -
- National Association of Medical Examiners, 2001 -
- Michigan Association of Medical Examiners, 2004 -
- American Society of Clinical Pathologists, 2002 -
- College of American Pathologists, 1998 - 2005
- Florida Association of Medical Examiners, 2001 - 2005
- American Medical Association, 2001 - 2005
- Florida Medical Association, 2001 - 2004
- Hillsborough County Medical Association, 2001 - 2004
- Florida West Coast Association of Pathologists, 2002 - 2004

IN THE UNITED STATES DISTRICT COURT
FOR THE NORTHERN DISTRICT OF IOWA
(Central Division)

LARRY ZUBROD, Individually and as Administrator for the ESTATE OF MICHAEL ZUBROD and CHERYL ZUBROD, Individually and as Administrator for the ESTATE OF MICHAEL ZUBROD, Plaintiffs, vs. SHAYNE HOCH, ISAAC SHORT, and JOHN SMITH, in each man's individual capacity as a Law Enforcement Officer for the Worth County Sheriff's Office; SHERIFF JAY LANGENBAU, in his individual capacity, and WORTH COUNTY, IOWA, Defendants.	Case No. 6:15-cv-02065-LRR REPORT OF DANIEL J. SPITZ, M.D. REGARDING TASER USE ON DECEDENT MICHAEL ZUBROD

AFFIDAVIT OF DANIEL J. SPITZ, M.D.

Daniel Spitz, M.D. hereby declare as follows:

1. I am a forensic pathologist with board certification by the American Board of Pathology in Anatomic, Clinical and Forensic Pathology. I currently serve as the Chief Medical Examiner for Macomb and St. Clair Counties in Michigan. I have personally performed over 5000 autopsies.

2. I have the following relevant experience and credentials which are detailed in my curriculum vitae (see attached).

3. I have experience opining as to cause of death in persons subjected to an ECD prior to death. I have reviewed/participated in approximately 8-10 death cases in which the use of force included a TASER.

4. As a result of my training and professional experience, I possess the

requisite knowledge of medical causation to certify the cause of death in a variety of situations.

5. It is well-known that application of a TASER causes pain and loss of voluntary muscle control.

6. As a result of my experience, knowledge and training, it is my expert opinion that TASERs are capable of causing and contributing to death in persons who are under the influence of stimulants as was the circumstance in this case.

7. As a result of my experience, knowledge and training, it is my expert opinion that TASERs are capable of causing and contributing to death when persons are subjected to multiple and/or prolonged bursts of electrical current.

8. Based on my knowledge, experience, education and training, I believe Michael Zubrod's death was caused by multiple factors, one of which was the Defendant Officers' repeated discharge of their TASERs into the decedent. I base my opinion on the fact that TASERs can cause pain, physical stress, psychological stress and in some cases cardiac arrhythmias and respiratory compromise.

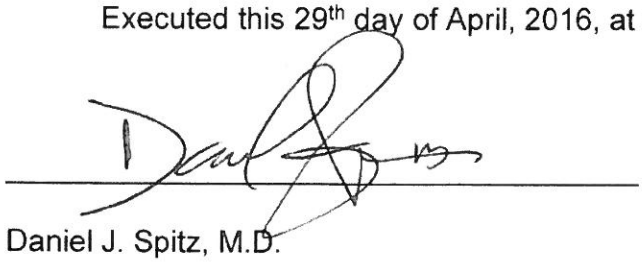
9. It is well known that a TASER discharge affects the breathing and heart rate of a subject which, when used repeatedly, can lead to detrimental health situations. Repeated and/or prolonged use of a TASER can result in respiratory distress by causing loss of normal muscular control.

10. I also base my opinion in this case, in part, on the conclusions reached by the State of Iowa Associate Medical Examiner, Dr. Jonathan Thompson, whose report indicates that the manner of death was homicide.

11. My opinion is further based on all of the cases in the public realm in which the application of a TASER was a causative or contributing factor in an individual's death, and on the hundreds of articles in the public domain linking TASER ECD exposure to an increased risk of death.

I declare under penalty of perjury, under the laws of the United States of America, that the foregoing is true and correct.

Executed this 29th day of April, 2016, at Clinton Township, Michigan.



Daniel J. Spitz, M.D.

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Electronic Control Devices: Science, Law, and Social Responsibility

Running title: *Myerburg et al.; Electronic control devices*

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The use of deadly force by law enforcement personnel is a hot-button issue in contemporary society. Their responsibility is to handle life-threatening situations while so far as possible avoiding fatalities or serious injuries of suspects, bystanders, or themselves. In these settings, the use of lethal weapons must be weighed against reactions to circumstances that do not pose an imminent threat of a fatal outcome. Moreover, use of intense physical restraint instead of lethal weapons in less ominous situations is not without risk either, in that fatal events due to asphyxia have occurred as an unintended consequence of this "non-lethal" control strategy. Moreover, the decision whether deployment of a lethal weapon, as opposed to physical restraint, is the proper action in a specific confrontation often must be made quickly, leaving little time for assessing options.

The development of electronic control devices (ECDs), or stun-guns, emerged as an alternative strategy, allegedly effective for controlling a threatening confrontation without resorting to needless serious injuries or fatalities. In principle, the ECD is more forgiving of errors of judgment than a firearm. The ECD is designed to deliver a painful and physically disabling electric shock, temporarily incapacitating a threatening individual. Although a significant number of deaths in association with, or following the use of, ECDs have been cited in a lay publication,[1] peer-reviewed literature on the topic has been limited to several case reports, largely in the form of letters to the editors of journals.[2] However, associations or correlations, in the absence of extremely strong population-based hazard ratios,[3] are inadequate to support a scientific conclusion of causation, without additional information supporting the likelihood of a direct causal relation in an individual case. Data limited to a few case reports makes it difficult to provide informed statements about safety and appropriate precautions.

New Information

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In this issue of *Circulation*, Zipes provides observational data from a series of 8 cases in which a reasonable accumulation of information on surrounding circumstances, clinical and pathologic data, and documented arrhythmias is provided.[2] The source of his data is unique, as it is derived from information provided to him in his disclosed role as an expert during litigation of these cases. Based upon his analysis of the data, Zipes opines that the data from the cases he analyzed, in conjunction with previously published clinical and experimental observations, suffices to support a conclusion that ECD shocks, in some circumstances, can cause electrical capture of the heart and initiate cardiac arrest. He does not conclude that these cases reveal a fundamental flaw in the design of the devices.

Analysis of the Data

The most compelling elements in the data provided are that in each of the reported cases, loss of consciousness (LOC) occurred during the application of an initial or subsequent shock, and that in at least 6 cases the initial rhythm recorded was ventricular tachycardia/fibrillation. Since LOC under the reported conditions does not necessarily equate with cardiac arrest, the implications of these observations are diluted to some degree by the inability of the device to record the rhythm at the time of LOC, and by the lack of data that uniformly establishes the absence of a pulse immediately upon LOC. In 3 cases, the subjects were observed to be breathing after LOC, and two of these had a pulse initially. The observation of "breathing" does not, however, exclude cardiac arrest, since "gasping" respirations can occur during cardiac arrest and be confused with true breathing by lay observers.[4] In addition, blood alcohol levels >0.30 gm/100 ml were recorded in 2 subjects; such levels themselves may be associated with LOC and, occasionally, death.

The source of the data leads to some concerns about distortions and biases that can

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develop during the adversarial litigation process; but overall there is enough objective data to support reasonable judgments in the individual cases, if not definitive conclusions generalizable to all cases. Based on the circumstances, timing, and rhythm strips provided, and the pathologic data provided, it seems reasonable to conclude that some finite number of these cases, greater than zero, but likely less than all 8, demonstrates a direct association between delivery of an ECD shock and the onset of cardiac arrest in an individual in whom other possible causes are not present. One of the problems in interpreting the data is that there were undisputed pathologic findings of a normal heart in only 2 of the 7 autopsied fatal cases, with a mildly elevated heart weight and a blood alcohol level of 0.25 gm/100 ml in one of these 2. In both of these cases, the descriptions of the incidents and supporting data lend credence to the likelihood of an association that is strong enough to demonstrate a cause-and-effect relationship.

In the remaining cases, various levels of uncertainty result from the description of associated pathological findings; and in some cases, disparate opinions were offered by pathologists on opposing sides of the cases. In addition, in 2 cases, the delay between collapse and loss of pulse is consistent with LOC due to causes other than direct induction of ventricular fibrillation or pulseless ventricular tachycardia by the ECD shock, since a direct induction of a pulseless arrhythmia would likely be immediate. Certainly, an initial hemodynamically-stable ventricular tachycardia that subsequently deteriorated to a pulseless ventricular arrhythmia could not be excluded. These considerations limit the ability to establish independent causation in each case, but they are not sufficient to exclude an ECD shock as a possible cause or significant contributing factor to the induction of ventricular fibrillation consistently. Within the limits of the data provided for each of the cases, it seems fair to conclude that Zipes has provided sufficient information to amount to a proof of concept for a potential causal relationship between

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an ECD discharge and the initiation of ventricular fibrillation. The likelihood of a causal relationship in any individual case would have to be judged on the basis of the combined information available. This can be challenging, because it is often difficult to distinguish between etiologic pathology and bystander pathology when the circumstances of a death are complex.

Stress Physiology and Causation

The interpretation of causation is made more complex by the stressful nature of the circumstances in which an ECD is usually used. The stress response may be mediated by the confrontation itself, by pain from the delivered shock, or by a combination of the two. A stressor resulting in epinephrine release is associated with greater vulnerability to electrical induction of ventricular fibrillation,[5] in both normal and abnormal hearts. However, a stressor may also have adverse effects independent of its influence on responses to direct electrical stimulation of the heart. If epinephrine release is assumed to interact with a pre-existing pathologic abnormality in the causation of a cardiac arrest, it is difficult to determine whether the pathophysiological cascade leading to cardiac arrest is initiated by the confrontation or pain from the shock, as opposed to direct electrical stimulation of the heart. In 3 of the cases, there are contested diagnoses of hypertrophic cardiomyopathy, right ventricular dysplasia, and lymphocytic myocarditis, each of which, if present, can be associated with cardiac arrest during stress.

Transient stressors, other than ECD shocks, can be triggers for cardiac arrest. For example, on the day of the 1994 Los Angeles earthquake, the incidence of sudden cardiac death in the Northridge area was >5 times higher than the expected rate, as calculated from multi-year historical controls, and the incidence during the week following the earthquake was lower than expected.[6] This led to the conclusion that the stress of the earthquake advanced events about to

happen among people with coronary disease to a single point in time when a common stressor was shared by the population. Observations such as this further complicate the identification of causation in disparate, stress-associated cases. Accordingly, the cumulative experience and data available in the Zipes report, and in other reports, cannot be generalized to a conclusion of a fundamental flaw in the concept, design or properties of ECD devices that can be generally applied to all cases. Each case must be evaluated in light of all its facts and conditions, including proper use of the device, in order to arrive at an opinion on specific causation.

Pathophysiology and Biological Plausibility

There are arguments supporting the claim that, under appropriate circumstances, it is biologically plausible that an ECD discharge can be a direct trigger for ventricular fibrillation. Both clinical concepts and experimental data support this, and are well-summarized in Zipes' discussion.[2] The most salient points are that the energy delivered by the device is sufficient to achieve transthoracic capture when delivered to the anterior chest, analogous to clinical transthoracic pacing,[7] in combination with a rate of stimulation that is sufficient to induce ventricular fibrillation. The notion that epinephrine release enhances the ability to achieve capture is supported by experimental studies, but its significance is confounded by the presence of pre-existing disease which can also be influenced by epinephrine release. Published experimental studies support these clinical notions, and therefore the proof-of-concept conclusion. Clinical and experimental data indicating biological plausibility is among the factors supporting a transition from an observation of an association to acceptance of a conclusion of causation.[3]

Random Selection Bias versus Population Effect

The absence of any form of control group, such as a denominator providing numbers of ECD shocks delivered without potentially fatal consequences, and their corresponding circumstances,

complicates efforts to determine the magnitude of risk of adverse events. Unfortunately, the accumulation of such data, absent a registry of ECD discharges, is virtually impossible to collect retrospectively. Local or regional registries, if established in the future, could both provide denominators and correct any under-reporting of adverse events. It would be very helpful to be able to estimate the frequency of LOC without cardiac arrests under circumstances similar to these 8 cases, as well as the incidence of confrontation-associated cardiac arrests in these circumstances, absent the deployment of ECDs.

Transition from Population Science to Rules of Evidence in Law

Causation arguments in civil law cases employ the dual concepts of *general* and *specific* causation. The former refers to the claim that a substance, device or action has properties that *can* cause the adverse outcome in question, while the latter asks the question whether it *did so* in the specific case under consideration. The U.S. legal process usually requires demonstration of general causation before a specific causation claim can be argued.[8] Under the Federal Rules of Evidence,[9] as elucidated by the U.S. Supreme Court in Daubert v. Merrell Dow,[10] scientific reliability and relevance, as determined by judges and based on expert testimony during formal hearings on evidence, defines what can be placed before a jury. In some rulings, the absence of statistically significant population data has been held to be grounds for excluding general causation claims.[11] Questions of specific causation can become even more complex. A number of jurisdictions have accepted the notion that a relative risk greater than 2.0 is necessary and/or sufficient for proof of specific causation, most notably in toxic tort cases.[12] There are theoretical and practical reasons why this standard is flawed, not the least of which is the fact that it shifts *individual* specific causation to a *cumulative* metric of causation, which conceptually is more aligned with general causation than specific causation. Accordingly, the absence of any

population data on the ECD/cardiac arrest issue is a limiting factor for general causation claims, as well as specific causation based on the relative risk standard. This relegates specific causation theories to associations supported, but not necessarily determined, by factors such as temporal relationships, biological plausibility, and experimental evidence.[3,8] In addition, rulings prohibiting specific causation arguments, in the absence of accepted evidence for general causation, have not been uniform across all jurisdictions.

Lessons Learned From the ECD Data

Practical questions pertaining to the use of these devices by law enforcement officers derive from observations such as those reported.[2] In the absence of information that undermines the rationale for, and general usefulness of, these devices as an alternative to lethal weapons or potentially dangerous physical force, the use of these devices by law enforcement officers should not be stopped on the basis of the information provided in the report, as Zipes also notes. However, two policy and procedure considerations emerge very strongly from the report. One is that there has to be appropriate education of law enforcement officers as to the potential, however remote, for an adverse outcome, either directly or as a contributing effect in the presence of pre-existing cardiac abnormalities. This calls for educational efforts that emphasize the potential concerns about factors that law enforcement officers can control, such as prolonged and repetitive shocks and avoiding shocks to the anterior chest, when conditions permit. The second precaution is that officers should be educated to consider that any subject exposed to such a shock who loses consciousness should be assumed to be in cardiac arrest until proven otherwise. Responses to these circumstances must not be delayed. When AEDs are available in police vehicles,[13] they should be immediately deployed following LOC.

There remains an overarching ethical question about the proper uses of these devices.

If one assumes a very low risk of induction of ventricular fibrillation by ECDs, even with proper deployment and use according to instructions, then a distinction must be made between the use of these devices as a alternative to potentially lethal force and their use arbitrarily under circumstances in which physical interventions would be sufficient. Despite a low risk of the possibility of ventricular fibrillation as an unintended consequence of ECD use, their use seems appropriate to avoid the more likely fatal outcome with a lethal weapon. However, if ECDs are used indiscriminately under circumstances in which lethal weapons or intense physical force would not be necessary, then *any* risk of ventricular fibrillation induction would be unjustified. As with any lethal risk, the question whether use of ECDs is appropriate must be considered in light of the difficult and rapid judgments that must be made about the circumstances of individual cases. This ethical and practical challenge should be reflected in policies and procedures guiding law enforcement activities, and woven into the education of law enforcement officers who live with this responsibility daily.

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References:

1. USA: 'Less than lethal'? List of deaths following use of stun weapons in US law enforcement 3 June 2001 to 31 August 2008. Amnesty International Publications. Index Number AMR 51/010/2008. December 16, 2008.

2. Zipes DP. Sudden cardiac arrest and death associated with application of shocks from a TASER electronic control device. *Circulation*. 2012;125:XXX-XXX.

3. Hill AB. The environment and disease: Association or causation? *Proc Roy SocMed*. 1965;58:295-300.

4. Bobrow BJ, Zuercher M, Ewy GA, Clark L, Chikani V, Donahue D, Sanders AB, Hilwig RW, Berg RA, Kern KB. Gasping during cardiac arrest in humans is frequent and associated with improved survival. *Circulation*. 2008;118:2550-2554.

5. Verrier RL, Lown B. Behavioral stress and cardiac arrhythmias. *Annu Rev Physiol*. 1984;46:155.

6. Leor J, Poole WK, Kloner RA. Sudden cardiac death triggered by an earthquake. *N Engl J Med*. 1996;334:413-419.

7. Falk RH, Zoll PM, Zoll RH. Safety and efficacy of noninvasive cardiac pacing. A preliminary report. *New Engl J Med Sci*. 1983;309:1166-1168.

8. Haack S. Proving causation: The holism of warrant and the atomism of Daubert. *J Health Biomed Law*. 2008;4:253-290.

9. Act to Establish Rule of Evidence for Certain Courts and Proceedings, Pub. L. 93-595, 88 Stat. 1926.; see also Restyled Fed. R. Evid. 702 (2011).

10. Daubert v. Merrell Dow Pharmaceuticals, Inc., 509 US 579, 589 (1993).

11. In re: Ephedra Products Liability Litigation 04 MD 1598 (JSR), 393 F. Supp. 2d 181 (SDNY 2005).

12. Merrell Dow Pharmaceuticals, Inc. v. Havner 953 S.W.2d 706 (Tex. 1997).

13. Myerburg RJ, Fenster J, Velez M, Rosenberg D, Lai S, Kurlansky P, Newton S, Knox M, Castellanos A. Impact of community-wide police car deployment of automated external defibrillators on survival from out-of-hospital cardiac arrest. *Circulation*. 2002;106:1058-1064.

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Acute Effects of TASER X26 Discharges in a Swine Model

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Background: **Very little objective laboratory data are available describing the physiologic effects of stun guns or electromuscular incapacitation devices (EIDs). Unfortunately, there have been several hundred in-custody deaths, which have been temporally associated with the deployment of these devices. Most of the deaths have been attributed to specific cardiac and metabolic effects. We hypothesized that prolonged EID exposure in a model animal system would induce clinically significant metabolic acidosis and cardiovascular disturbances.**

Methods: **Using an Institutional Animal Care and Use Committee-approved protocol, 11 standard pigs (6 experimentals and 5 sham controls) were anesthetized with ketamine and xylazine. The experimentals were exposed to two 40-second discharges from an EID (TASER X26, TASER Intl., Scottsdale, AZ) across the torso. Electrocardiograms, blood pressure, troponin I, blood gases, and electrolyte levels were obtained pre-exposure and at 5, 15, 30, and 60 minutes and 24, 48, and 72 hours post-discharge. p values <0.05 were considered significant.**

Results: **Two deaths were observed immediately after TASER exposure from acute onset ventricular fibrillation (VF). In surviving animals, heart rate was significantly increased and significant hypotension was noted. Acid-base status was dramatically affected by the TASER discharge at the 5-minute time point and throughout the 60-minute monitoring period. Five minutes postdischarge, central venous blood pH (6.86 ± 0.07) decreased from baseline (7.45 ± 0.02; $p = 0.0004$). P_{CO_2} (94.5 mm Hg ± 14.8 mm Hg) was significantly increased from baseline (45.3 mm Hg ± 2.6 mm Hg) and bicarbonate levels significantly decreased (15.7 mmol/L ± 1.04 mmol/L) from baseline (30.4 mmol/L ± 0.7 mmol/L). A large, significant increase in lactate occurred postdischarge (22.1 mmol/L ± 1.5 mmol/L) from baseline (1.5 mmol/L ± 0.3 mmol/L). All values returned to normal by 24 hours postdischarge in surviving animals. A minor, nonsignificant increase in troponin I was seen at 24 hours postdischarge (0.052 ng/mL ± 0.030 ng/mL, mean ± SEM).**

Conclusions: **Immediately after the discharge, two deaths occurred because of ventricular fibrillation. In this model of prolonged EID exposure, clinically significant acid-base and cardiovascular disturbances were clearly seen. The severe metabolic and respiratory acidosis seen here suggests the involvement of a primary cardiovascular mechanism.**

Key Words: **Taser, Electromuscular incapacitation, Acidosis, Electrocardiograph.**

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Stun guns or electromuscular incapacitation devices (EIDs) generate between 25,000 and 250,000 V and can be discharged for as long as 5 to 10 minutes continuously. It is estimated that millions of people in the United States are at risk of exposure to EIDs daily and that thousands are exposed annually.[1] Amnesty International has compiled a list of more than 100 fatalities from cardiac arrest in the 2001 to 2004 period that were associated with EID use in the United States.[2] This growing list of fatalities has reinvigorated interest in the safety of EIDs and potential complications associated with their use, especially their ability to induce fatal ventricular dysrhythmia.[3–6] In the United States alone, more than 200,000 individuals have been exposed to discharges from the most common type of EID, the TASER. In training sessions, more than 24,000 law enforcement personnel have been exposed to brief TASER discharges but no deaths have been documented. Worldwide, TASERs are currently used by more than 9,100 law enforcement agencies and owned by more than 115,000 private citizens (www.taser.com). Despite the increasing usage of TASERs and other EIDs, there is no consensus in the biomedical community regarding their safety.

EIDs are very effective when used by law enforcement to incapacitate combative suspects while reducing the risk of injury to officers, suspects, and by-standers.[2] These devices utilize time-varying DC currents that evoke strong, repetitive contractions in most or all of the somatic musculature. The mechanism by which this occurs and the pathophysiologic effects of these discharges are poorly understood. There are several reasons for this lack of information including but not limited to (1) disagreement about the electrical output from these devices especially under resistive load, (2) disagree-

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ment about what load is appropriate or representative, (3) the lack of a standard model for study in vitro or in vivo, and (4) a general disinclination for academic scientists to study these devices because they are viewed as weapons.[3–5,7] Under no-load conditions, the TASER X26 delivers DC pulses at a voltage of about 50 kV, with a pulse duration of 140 μseconds, a frequency of 19 Hz, and power of 0.36 J/pulse (www.taser.com). In vivo, this type of discharge causes severe pain, strong muscle contractions, and incapacitation of volitional movement. However, Ruggieri[7] asserts that the peak currents achieved by the TASER under physiologic resistive loads can be many times higher than 2.1 mA and may easily exceed the ventricular fibrillation (VF) threshold. As a result of such disparities, the safety profile and effects of EID current exposure on the function and structure of living tissue cannot be extrapolated reliably.

Many of the initial studies on EIDs were performed on the much less powerful first or second generation devices.[8–10] The recent peer-reviewed literature on fourth generation EIDs (such as the TASER X26) is still emerging and the results are conflicting. Some studies, using a TASER-like device, showed no evidence of acute dysrhythmia and a large safety margin for the development of ventricular dysrhythmia in swine.[4,11] Similarly, neither acidosis nor hyperkalemia have been observed in healthy human volunteers exposed to brief (2.7 seconds on average) TASER X26 discharges.[12,13] However, in swine models, Jauchem et al.[14] showed the development of significant acidosis, and Webster et al.[15] and Nanthakumar et al.[16] have shown the potential for fatal dysrhythmias with TASER X26 exposure. Such conflicting results make it difficult to establish guidelines regarding the need for treatment or even monitoring of the increasing number of patients who arrive in emergency rooms after exposure to EID discharges.

In an attempt to reconcile some of the conflicting information about the effects of TASER discharges, we have studied the effects in a well-characterized swine model. Our working hypothesis was that subcutaneous discharges from the TASER X26 can produce significant cardiac effects including acute dysrhythmia or VF and that these effects may be exacerbated by concomitant acidosis or electrolyte and biochemical abnormalities.

MATERIALS AND METHODS

Animals and Groups

Three- to 6-month-old Yorkshire pigs (Michael Fanning Farms, Howe, IN) weighing between 22 kg and 46 kg were used. The experimental group (80-second thoracic discharge) and the negative or sham control group, were comprised of six and five animals, respectively. The size of the animals used in our study correlates with that of children, teenagers, and some adult humans with small frames. Other investigations of TASERs have used animals in a similar size range.[4,11,14,16] The Institutional Animal Care and Use Committee (IACUC) for the Hektoen Institute for Medical Research, LLC reviewed and approved this project.

All animals were deeply anesthetized during each monitoring session using intramuscular and intravenous ketamine (Ketaset; Fort Dodge Animal Health, Fort Dodge, IA) and xylazine (Anased; Lloyd, Shenandoah, IA), and respiratory secretions were inhibited using glycopyrrolate (Robinul; Fort Dodge Animal Health, Fort Dodge, IA). Ketamine/xylazine/glycopyrrolate (30/3/0.01 mg/kg) was administered intramuscularly for sedation and then ketamine and xylazine (5.6/0.8 mg/mL) in sterile saline were instilled intravenously using an infusion pump (Flogard 6200; Travenol, Deerfield, IL) through a 23-guage cannula placed into an ear vein at a rate of 3 $mL \cdot h^{-1} \cdot kg^{-1}$ (16.8/2.4 mg/kg). Animals were intubated using cuffed endotracheal tubes (5.0–6.5 mm, Rusch; Kernen, Germany) after anesthetizing the larynx with 0.25 mL to 1.0 mL of sprayed 20% benzocaine (Hurricaine; Beutlich Pharm., Waukegan, IL). Breathing was controlled (15 breaths per minute; tidal volume = 10 mL/kg; minute volume = 150 mL/kg). The TASER X26 was discharged (see below) in two separate 40-second intervals for a total of 80 seconds, during which time the ventilator was shut off but spontaneous breaths were permitted. Two ventilated breaths (within 10 seconds) were administered between the 40-second discharges. Breathing rate was adjusted after discharge according to demand. The purpose of this was to ensure that the ventilator was not the cause of any observed respiratory acidosis. Animals were maintained in dorsal recumbence for all electrical discharges and monitoring procedures. At the conclusion of each monitoring session, intravenous yohimbine (0.05–0.15 mg/kg; Yobine; BenVenue Labs, Bedford, OH) was used to reverse the effects of xylazine and to speed recovery from anesthesia.

Instead of using inhaled halothane or isoflurane anesthesia, ketamine/xylazine was used throughout this study (except for thoracotomized animals). The primary local electrical injury anticipated with these waveforms was membrane electroporation, particularly of nerve and muscle.[17,18] This effect is sensitive to the presence of lipids or highly lipid-soluble agents such as isoflurane, halothane, or barbiturates. The ketamine and xylazine combination used here has also been shown to be an effective general anesthetic in swine[19,20] and our data confirm this (see below).

Test Device

An unmodified, police-issue TASER X26 device was used to produce electromuscular incapacitation. Because it is illegal for civilians to possess the TASER X26 in Illinois, a member of the local law enforcement community trained in TASER use delivered the discharges. TASER lithium 6 V Digital Power Magazines (DPM) were used as the power source for all discharges. DPM charge state was monitored before and after each discharge and at no time was a DPM used with a charge state less than 70%.

Experimental Set-Up and EID Discharge

While in dorsal recumbence, all four limbs of the animal were restrained to the table. The TASER cartridge was fired

into a towel and the darts were disentangled from the cloth without disrupting any of the fine wires, insulation, or connections. The barbed darts were placed along a line parallel to the cardiac axis. The superior or noncurrent-emitting dart was placed 13 cm superior to the xyphoid process and 5 cm to the right of the midsternal line. The lower or current emitting dart was placed 7 cm to the left of the umbilicus. This dart configuration produced a diagonal separation of approximately 30 cm in each animal and is similar to that used by Jauchem et al.[14] For two of the six animals in the experimental group, the superior dart was the current emitting dart. All darts were manually inserted perpendicular to the skin and to the maximum depth allowed by the length of the barbed end (3/8 of an inch) such that the dart tip was located in subcutaneous tissue. For TASERs discharged from distances less than 11 feet, skin penetration has been shown to occur for both darts in approximately 65% of TASER strikes in the field.[21]

The TASER X26 was discharged in two separate 40-second intervals for a total of 80 seconds, during which time the ventilator was shut off but spontaneous breaths were permitted. Two ventilated breaths were administered during the 10-second pause between the 40-second discharges. The discharge times of stun devices as used in the field vary greatly. Often short bursts (~5 seconds) are sufficient to subdue most subjects, but the devices are capable of delivering very prolonged, continuous discharges. The only practical limit on the discharge duration is the amount of battery power available, so continuous discharges could be administered for more than 10 minutes and instances of discharges longer than 90 seconds have been reported for TASERs.[22] Subjects are usually incapacitated almost immediately upon exposure to TASER discharges but they regain muscle control very rapidly after discontinuation of the discharge. Highly combative subjects may receive prolonged or repeated discharges, during which time officers can approach and restrain them.

Cardiac Rhythm and Echocardiography

Cardiac rhythm was evaluated and monitored continuously during anesthesia using a five-lead electrocardiogram (EKG) and monitor (Datex instruments, Helsinki, Finland) and at each experimental time point 10- to 15-second tracings were printed and retained. EKGs were also recorded throughout the duration of the discharge. Because of the amount of electrical interference created by the TASER discharge, EKGs done during the discharge were unreadable. To adequately assess the rhythm and function of the myocardium, echocardiography was performed using a Sonosite 180 with a 2-MHz probe (Sonosite Inc. Bothell, WA) on four of the six experimental animals. Echo images were first obtained predischarge to establish a baseline for each animal in the left parasternal axis. Echocardiography was then continued during and after the TASER discharge to assess, in real time, any changes that occurred in myocardial rhythm and function. Video records of each echo were digitally recorded for further analysis.

Controls

Five sham control animals were studied for 72 hours using the same paradigm as that used for animals exposed to TASER discharges except that they were not exposed to any discharges during the monitoring period. At the completion of the 72-hour blood sampling and monitoring period, two of these sham animals underwent thoracotomy. Each of these animals then received two 40-second TASER discharges while direct visual monitoring was performed. Each of these animals was physiologically normal before these discharges according to all blood chemistries, vital signs, and EKG. These animals had received no previous TASER discharges. In addition, baseline intra-animal data were obtained for all 11 (6 experimentals and 5 sham controls) animals studied.

Thoracotomy

To further document cardiac activity, two 40-second discharges were administered to two of the five control animals (31 kg and 46 kg) just before being euthanized (see above). Left anterior thoracotomies were performed under inhaled anesthesia with 1.5% to 2% isoflurane. Electrocautery was avoided to eliminate any non-EID electrical exposure. An incision approximately 10-cm long was made over the left anterior thorax in the fifth or sixth intercostal space. Sharp dissection was carried down to access the left thorax. A rib spreader was used to expose the heart and lungs. The rib spreader was placed outside the current path between the darts and it was not in direct contact with the heart. The left lung was retracted out of the field with gauze sponges. The pericardium was opened sharply facilitating a direct view of the beating myocardium. The TASER darts were then placed in the manner previously described and two 40-second discharges were administered. The 31-kg animal received the second 40-second discharge with the superior dart as the current emitting dart. Cardiac activity was directly monitored and recorded before, during, and after TASER discharge for subsequent analysis and comparison with echocardiographic data.

Blood Samples and Analysis

There were eight time points at which central venous blood was drawn from the precaval venous complex, and vital signs (tissue oxygen saturation, heart rate, and blood pressure [BP]), and additional EKGs were recorded. The sampling time points included predischarge (time 0) and 5, 10, 15, 30, 60 minutes, 24, 48, and 72 hours postdischarge. Animals were euthanized according to American Veterinary Medicine Association standards after the 72-hour time point by switching the anesthesia to 5% inhaled isoflurane and injecting 3 mol/L KCl into the heart.

Immediately after being drawn, each blood sample was placed into heparinized and plain vacutainer tubes. The heparinized blood was tested using an iSTAT analyzer (Abbott Point-of-Care, Abbott Park, IL) using CG8+, CG4+, creatinine, and troponin I (TnI) cartridges. These cartridges return

data on pH, P_{CO_2}, bicarbonate, lactate, potassium, TnI, and creatinine. Blood samples were stored on ice for a maximum of 2 hours, centrifuged (3,000*g* for 15 minutes at 4°C), plasma and serum aliquoted into 400 μL microcentrifuge tubes, and samples stored at -85°C until use. Serum from each time point was thawed and assayed for creatine kinase-MB isoform (CK-MB) and myoglobin using microplate enzyme-linked immunosorbent assays.

When whole blood lactate values exceeded the CG4+ maximum value of 20.0 mmol/L, the aliquoted serum was diluted 1:1 with normal saline (0.9% NaCl). The diluted serum was then assayed using a CG4+ cartridge to get a numerical lactate value. This value was then doubled and entered into the data set. This dilution method was validated by first diluting iSTAT standards in a similar fashion and analyzing them using a CG4+ cartridge. The values obtained for the diluted iSTAT standards were one-half of the values expected for undiluted standards. The manufacturer has validated the use of serum for lactate determinations instead of whole blood.

Serum Myoglobin and CK-MB Determination

Plasma or serum myoglobin, TnI, and CK-MB have been shown to be useful in evaluating cardiac muscle damage because of myocardial infarction.[23–27] The time course for the appearance of each of these markers is known. Levels of cardiac TnI, the most specific marker for myocardial damage, peak at 12 to 24 hours, and may remain elevated for several days. Serum myoglobin becomes elevated within 2 to 4 hours of myocardial injury. CK-MB is found in cardiac and skeletal muscle but is present in much higher quantities in cardiac muscle. CK-MB levels become elevated within 3 to 4 hours of cardiac injury and remain elevated for 60 to 70 hours. Myoglobin and CK-MB can become elevated from noncardiac related injuries such as chronic muscle disease, skeletal muscle trauma, and renal failure.[23,28,29] As a result, it is common to evaluate all three of these markers to determine the extent of cardiac and skeletal muscle injury.

Serum samples stored at −85°C were thawed once and tested for myoglobin (20 μL/well) and CK-MB (25 μL/well) using solid phase microplate sandwich enzyme-linked immunosorbent assays (Diagnostic Automation, Calabasas, CA). All samples and standards for these assays were performed in duplicate and averaged. Standard curves using four to seven reference standards of different concentrations were generated for each run. Myoglobin and CK-MB concentrations for the experimental serum samples were interpolated from these standard curves using best-fit regression formulas generated by Excel (Microsoft, Redmond, WA).

Data Reduction and Statistical Analysis

All data points represent means ± SEM for each parameter. Parametric statistics including two-way analysis of variance (ANOVA) or paired *t* tests were used to compare quantitative data and groups. Trends were evaluated using linear regression. The experimental groups were compared against their own baseline and against the control group for each parameter (Prism v.3.03, GraphPad Software, San Diego, CA). Vital signs and blood chemistry values obtained for the animals that died within minutes of TASER discharge were not included in the statistical analyses.

RESULTS

Vital Signs Were Severely Altered by TASER Discharge

No spontaneous respiratory effort was observed during TASER discharge. An acute onset of tachycardia was noted after TASER discharge. Heart rate increased from a baseline of 103 bpm ± 9 bpm (mean ± SEM). The heart rate was greatest at the 5 minutes postdischarge time point (157 bpm ± 5 bpm; $p = 0.0085$ vs. baseline). Heart rate then gradually decreased during the remainder of the 60-minute monitoring period, but was not observed to return to the baseline until the 24-hour time point. At the 24-hour time point and subsequent time points, heart rates in experimental animals were similar to those of controls. The acute onset of tachycardia was not seen in control animals. Control animals showed a decrease in heart rate from baseline (91 bpm ± 2 bpm) during the initial 60-minute monitoring period with a nadir at 60 minutes (72 bpm ± 2 bpm; $p < 0.05$ vs. baseline). The observed effect on heart rate in the experimental group was significantly different from that of the control group when compared for the initial 60-minute monitoring time period by two-way ANOVA ($p < 0.0001$).

BP (Fig. 1) showed a decrease after TASER discharge in the first 60 minutes. BP reached a nadir at 15 minutes postdischarge (systolic BP = 79 mm Hg ± 8 mm Hg) in the experimental group. This decrease in systolic BP was significant ($p = 0.02$) compared with the baseline value (133 mm Hg ± 8 mm Hg) in the experimental group. The systolic BP gradually increased during the 60-minute monitoring period and returned to baseline values at 24 hours. BP did not show any significant changes in the control group. The difference observed between controls and experimental BP was significant (two-way ANOVA; $p < 0.001$).

One Experimental Animal Died of Acute VF

One animal in the experimental group (29 kg) died from VF after TASER discharge. Cardiac rhythm could not be discerned by EKG during the discharge because of the electrical interference and muscle contractions created by the TASER. Cardiac rhythm was evaluated by echocardiography during the discharge and found to be consistent with ventricular tachycardia. When the discharge ceased, sustained ventricular tachycardia was noted on echocardiography and confirmed by EKG (Fig. 2). During the course of the next few minutes, the ventricular tachycardia then degenerated into fatal VF. As previously indicated, all surviving experimental animals showed brief atrioventricular (AV) dyssynchrony followed by sinus tachycardia

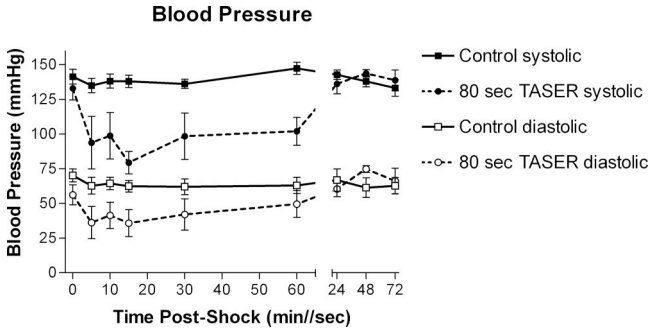

Fig. 1. *Blood pressure versus time for the 72-hour time course after TASER discharge. Both systolic and diastolic pressures (mean ± SEM) are plotted for the experimental and control groups. Systolic blood pressure reached a nadir at* t = *15 minutes (79 mm Hg ± 8 mm Hg). This value was significantly different from experimental baseline (*p = *0.02) and from control values at* t = *15 minutes (*p < *0.001). The differences observed between control and experimental blood pressures at all time points were significant (two-way ANOVA;* p < *0.001).*

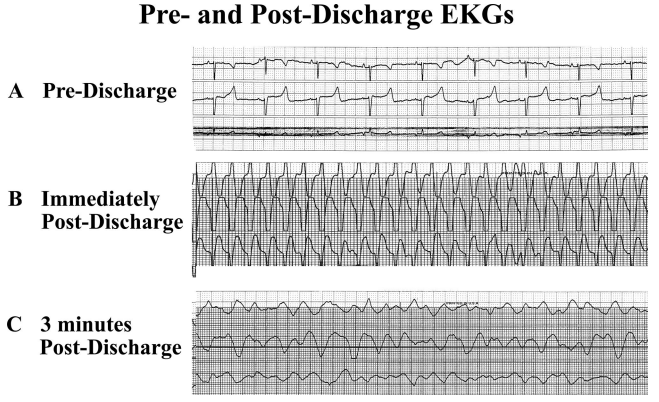

Fig. 2. *EKGs from one animal taken before (*A*) and after TASER discharge showing sustained ventricular tachycardia immediately after the discharge (*B*) followed by VF approximately 3 minutes later (*C*).*

after the discharge. Despite persistent sinus tachycardia, no EKG evidence of acute dysrhythmia was seen in the surviving animals.

Echocardiography (echo) showed capture of the ventricular rhythm during TASER discharge but motion artifacts prevented quantitative analysis of cardiac output and ejection fraction. One animal, as described above, went into VF after the discharge as confirmed by EKG and echo. The remaining three animals all showed capture of ventricular rhythm with rapid ventricular contractions seen on echo consistent with ventricular tachycardia (approximate rate of 300 bpm). This capture of cardiac rhythm occurred immediately after the start and continued for the duration of the TASER discharge as seen by echo. Sinus rhythm was regained after a brief period of AV dyssynchrony in each of these three animals and sinus tachycardia began within 1 minute after termination of the discharge.

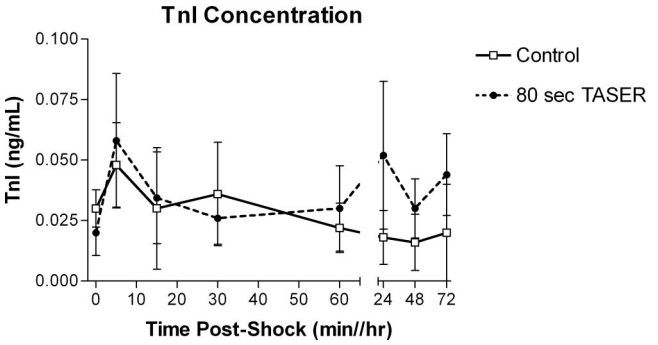

Fig. 3. *Troponin I (TnI) values during the 72-hour time course after TASER discharge. TnI values showed an initial increase from baseline values at 5 minutes postdischarge in both experimental and control animals. No significant differences were seen when TnI values for experimental and control animals were compared using two-way ANOVA (*p > *0.05).*

TnI (Fig. 3) showed an initial increase from baseline in the experimental animals at the 5-minute time point. A similar increase was also noted in control animals. TnI levels peaked at 24 hours postdischarge (0.052 ng/mL ± 0.03 ng/mL), this was not a significant increase from baseline values (0.02 ± 0.01, $p > 0.05$). No significant differences were seen when TnI values for experimental and control animals were compared using two-way ANOVA ($p > 0.05$). No significant changes were seen in CK-MB at anytime compared with that of controls.

Severe Metabolic and Respiratory Acidosis was Seen After TASER Discharge

Central venous blood pH (Fig. 4) showed a large decrease from baseline (7.45 ± 0.02) after the TASER discharge at the 5-minute time point (6.81 ± 0.07; $p = 0.0004$).

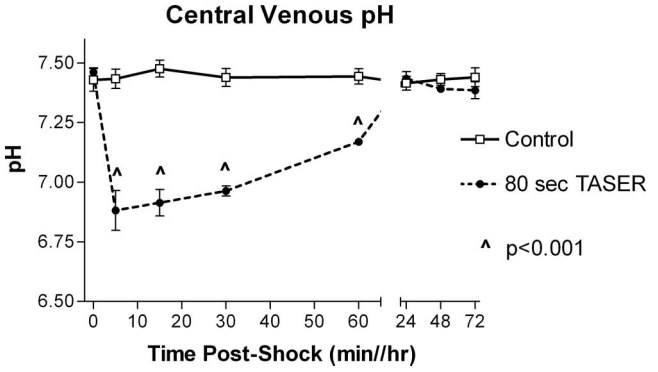

Fig. 4. *Central venous pH over time for control and experimental groups. Control animals had a baseline similar to experimentals but showed no significant changes from this baseline during the 72-hour monitoring period. A large decrease in central venous pH was noted after TASER discharge in the experimental group. Central venous blood pH was decreased throughout the 60-minute postdischarge monitoring session (*p̂ < *0.001, paired* t *tests) but returned to baseline values subsequently.*

In the experimental group, central venous blood pH decreased throughout the 60-minute postdischarge monitoring session but returned to baseline values subsequently. Control animals had a similar baseline and showed no significant changes during the 72-hour monitoring period. The observed difference was significant when compared using two-way ANOVA during the initial 60 minutes ($p < 0.001$).

Extreme hypercapnia was noted after TASER discharge (Fig. 5). A dramatic increase in P_{CO_2} was seen at 5 minutes (108.3 mm Hg ± 14.6 mm Hg) postdischarge. This change was in stark contrast to baseline values (45.3 mm Hg ± 2.6 mm Hg; $p < 0.0048$) for this group. The P_{CO_2} gradually decreased during the 60-minute monitoring period, and returned to baseline at subsequent time points. Control animals had normal P_{CO_2} values throughout the entire monitoring period. The difference observed between controls and experimental animals was significant (2-way ANOVA; $p < 0.001$).

Bicarbonate levels (Fig. 6) were found to be acutely and severely decreased from baseline values (30.8 mmol/L ± 0.9 mmol/L) at 5 minutes postdischarge (15.7 mmol/L ± 1.0 mmol/L; $p = 0.0046$). This decrease contrasted with the control group, which showed no significant changes in bicarbonate levels during the entire experimental time course. Bicarbonate levels remained decreased throughout the initial 60-minute postdischarge monitoring period and returned to baseline subsequently. When compared with controls over time, the observed changes in bicarbonate were significant (2-way ANOVA; $p < 0.001$).

Lactate values (Fig. 7) increased more than 13-fold after TASER discharge. Lactate levels increased from the experimental baseline of 1.6 mmol/L ± 0.3 mmol/L to 22.1 mmol/L ± 1.5 mmol/L ($p < 0.0001$) at 5 minutes postdischarge. Lactate levels remained elevated throughout the initial 60-minute monitoring period and returned to baseline values at 24 hours postdischarge.

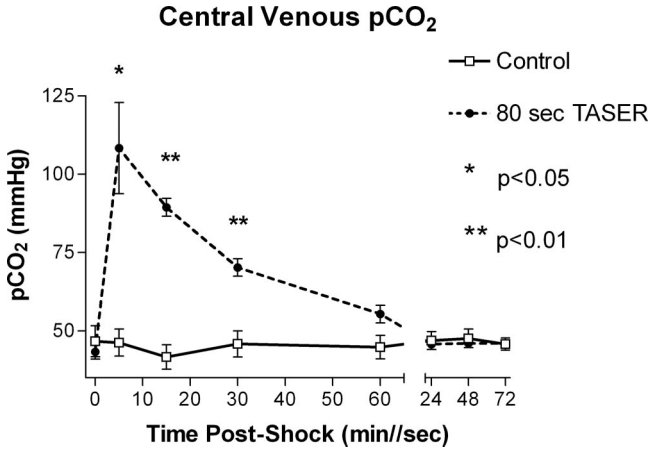

Fig. 5. *Central venous P_{CO_2} during the 72-hour time course after TASER discharge. A massive increase in P_{CO_2} was seen postdischarge (*p < 0.05, **p < 0.01, paired t tests). The P_{CO_2} gradually decreased during the 60-minute monitoring period and returned to baseline subsequently. Control animals had normal P_{CO_2} values throughout the entire monitoring period.*

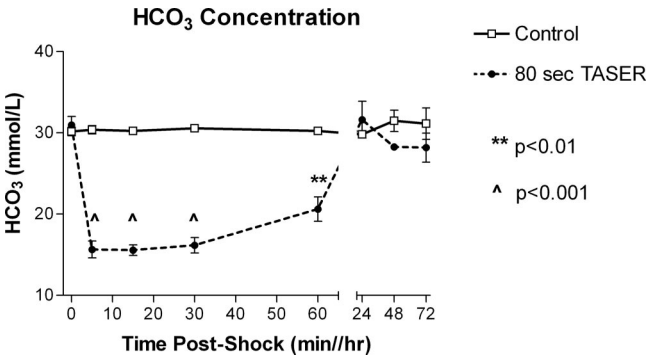

Fig. 6. *Bicarbonate concentration during the 72-hour time course after TASER discharge. The control group showed no significant changes in bicarbonate levels during the experimental time course. However, in the experimental group, bicarbonate levels were greatly decreased from baseline values (30.8 mmol/L ± 0.9 mmol/L) at 5 minutes postdischarge (15.7 mmol/L ± 1.0 mmol/L; p = 0.0046, t test). These levels remained decreased throughout the initial 60-minute postdischarge monitoring period and returned to baseline subsequently. When compared with controls over time, the observed changes were significant (two-way ANOVA; p < 0.001). Time points at which experimental and control values differed significantly are shown (**p < 0.01, ^p < 0.001, paired t tests).*

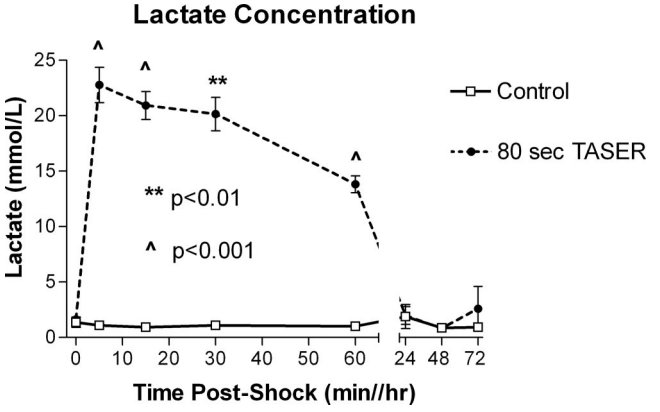

Fig. 7. *Lactate concentration during the 72-hour time course after TASER discharge. Control animals showed no significant changes in lactate levels, but these values increased more than 13-fold after TASER discharge in the experimental group. Lactate levels remained elevated throughout the initial 60-minute monitoring period and returned to baseline values at 24 hours postdischarge. Time points at which experimental and control values differed significantly are shown (**p < 0.01, ^p < 0.001, paired t tests).*

Control animals did not show any significant changes in lactate levels. Lactate levels in the control and experimental groups were significantly different when compared for the initial 60-minute monitoring time period (2-way ANOVA; $p < 0.001$).

Central venous oxygen saturation (Fig. 8) for the control and experimental groups was not significantly different at time 0 ($p = 0.70$). However, it decreased significantly after TASER discharge from the experimental baseline of 78.8% ± 4.6% at time 0 to 50.5% ± 5.8% at 15 minutes postdischarge ($p = 0.02$).

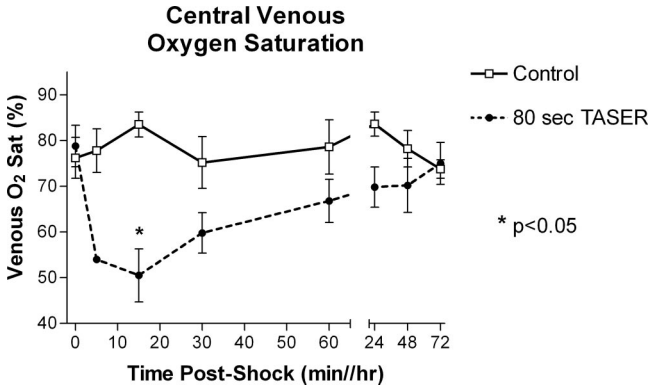

Fig. 8. *Venous oxygen saturation during the 72-hour time course after TASER discharge. Central venous oxygen saturation decreased significantly after TASER discharge and remained low throughout the initial 60-minute monitoring period. For the entire 60-minute postdischarge period, venous saturation for the experimental group was significantly decreased compared with that for controls (two-way ANOVA;* $p < 0.001$*). Time points at which experimental and control values differed significantly are marked (*$p < 0.05$*). Values returned to predischarge levels at 24 hours and remained at these levels subsequently.*

At 5 minutes postdischarge, venous oxygen saturation for only one sample (54%) was available in the experimental group. Other samples tested at this time returned "not measurable" results, likely because of the severe acid-base disturbance. As a consequence, this time point was excluded from the comparisons. Venous oxygen saturation remained low throughout the initial 60-minute monitoring period. Values for the experimental group then returned to predischarge levels at 24 hours and remained at these levels subsequently. For the entire 60-minute postdischarge period, venous saturation for the experimental group was significantly decreased compared with that for controls (2-way ANOVA; $p < 0.001$).

Thoracotomy

Before TASER discharge, with the heart exposed via left anterolateral thoracotomy (see supplemental video clip, ArticlePlus, www.jtrauma.com), normal sinus rhythm was directly visualized and confirmed by EKG. When the TASER discharge started, sinus rhythm was immediately (within 1 second) disrupted. In the 31-kg animal, the first 40-second discharge resulted in immediate capture of the myocardium producing rapid ventricular contractions consistent with ventricular tachycardia. During this discharge, atrial standstill was also seen. When the first discharge ceased, approximately 15 seconds of dyssynchronous atrial and ventricular contractions were noted, after which sinus rhythm resumed. During the period between discharges, the current emitting dart was relocated to the superior position. The second discharge resulted again in immediate disruption of sinus rhythm and ventricular tachycardia. Atrial standstill was again noted during this discharge. However, after 16 seconds, the ventricular tachycardia was replaced by fatal VF.

In the 46-kg animal, both 40-second discharges were administered with the TASER darts in the usual positions described in Methods. The first discharge resulted in immediate capture of ventricular rhythm resulting in ventricular tachycardia. Normal atrial contractions were noted during the discharge, but these contractions were not synchronized with ventricular contractions. When the discharge ceased, sinus rhythm resumed immediately. Similar cardiac effects to those seen in the first discharge were observed during the second 40-second discharge. At the end of the second discharge, sinus rhythm resumed immediately and sinus tachycardia was noted. This animal survived for 20 minutes without apparent ill effects at which time it was euthanized. For both animals, the cardiac activity directly visualized by thoracotomy was consistent with that seen by echocardiography.

TASER Discharge had Moderate Affects on Potassium, Sodium, and Creatinine Levels

Potassium values increased slightly in all animals from baseline to 5 minutes postdischarge. This increase was seen in both controls and experimental animals. The observed increase in potassium concentration at 5 minutes (4.2 mmol/L $\pm$ 0.1 mmol/L) in experimental animals was significant when compared with baseline (3.7 mmol/L $\pm$ 0.1 mmol/L). Potassium levels in the control and experimental groups were significantly different ($p = 0.0328$), but the differences were not clinically significant. At no time point did potassium values fall outside the normal range in any of the animals.

Creatinine values did not change significantly after TASER discharge nor did they exceed normal levels in any of the experimental animals (range, 1.1–1.7 mg/dL). Sodium levels showed an acute increase at 5 minutes after TASER discharge. The sodium concentration increased from a baseline value of 141.0 mmol/L $\pm$ 1.2 mmol/L to 148.2 mmol/L $\pm$ 1.6 mmol/L ($p = 0.0052$) then gradually returned to baseline at the 60-minute time point. Control animals showed a small decrease from baseline (138.3 mmol/L $\pm$ 4.8 mmol/L) at 5 minutes postdischarge (135.7 mmol/L $\pm$ 3.8 mmol/L; $p > 0.05$) and a return to baseline at 60 minutes postdischarge. The observed difference between control and experimental animals in sodium concentration was significant when compared for the 60-minute postdischarge time period (2-way ANOVA; $p < 0.0001$).

TASER Discharge Moderately Affected Serum Myoglobin

Mean serum myoglobin levels in the experimental group at 30 minutes postdischarge (25.9 ng/mL $\pm$ 3.2 ng/mL, $p = 0.0082$) were elevated when compared with baseline (12.7 ng/mL $\pm$ 1.9 ng/mL). However, during all time periods, myoglobin levels in the control and experimental groups were not significantly different (2-way ANOVA; $p > 0.05$). All other values were within normal limits and variations were not of clinical significance.

DISCUSSION

Case reports, autopsies, and retrospective analyses have suggested that EID discharge may be associated with fatal dysrhythmias in humans, although the occurrence of this complication is rare.[3–6,30,31] The dart placement chosen for the present study, with the current path traversing the left thorax, may represent a worst-case type of configuration for cardiac consequences from an EID such as the TASER X26. Our results show that during TASER discharges with this transcardiac vector there is a highly reproducible capture of cardiac rhythm producing ventricular tachycardia. Postdischarge effects included AV dyssynchrony and sometimes fatal VF.

Echocardiography showed that cardiac rhythm was unmistakably affected during every TASER discharge studied. Rapid or immediate onset of atrial standstill, ventricular tachycardia, or VF occurred during these discharges. This study is the first to show the effects of the TASER X26 on the myocardium during thoracic discharges using a combination of echo, thoracotomy, and EKG. Our observations are in general agreement with those of Nanthakumar et al.[16] who showed, using intracardiac EKG monitoring, that an unmodified TASER X26 can capture myocardial rhythm resulting in high rates of ventricular stimulation and potential dysrhythmia.[16]

In two of eight animals exposed to TASER discharge, one with and the other without thoracotomy, the capture of cardiac rhythm and ventricular tachycardia were followed by VF and death. These animals had not been exposed to TASER discharges previously, showed no pre-existing electrolyte abnormalities and displayed no other physiologic abnormalities before the TASER discharges. The experimental conditions used for each of these animals differed somewhat. One animal had undergone a thoracotomy and had been anesthetized with inhaled anesthesia, whereas the other animal did not undergo thoracotomy and had been anesthetized with intravenous ketamine and xylazine. It could be argued that inhaled anesthesia reduces the threshold for VF[16,19] and thoracotomy provides an atypical, more direct current path to the heart; however, neither of these conditions existed in the second case and fatal VF was seen nonetheless. It is possible that VF is a direct result of the current vector in combination with cardiac capture during the vulnerable period of ventricular repolarization (T-wave). Stimulation of the myocardium during this period has long been recognized as a cause of sustained ventricular dysrhythmia and sudden death.[16,32] The frequency of the TASER wave form (19 Hz) makes it highly likely that one or more pulses will occur during T-waves even with brief discharges (1–5 seconds), yet sudden death is very rarely seen subsequent to TASER discharges in humans. The mechanism whereby these discharges capture cardiac function clearly requires further study.

McDaniel et al.[4] showed that the threshold for VF with EID discharges was directly proportional to body mass for animals ranging from 30 to 117 kg. They also reported that the output of their custom-built TASER-like device had to be increased by a factor of 15 to induce VF with a 5-second discharge in 30 kg swine. Our animals varied in mass from 22 to 46 kg and two animals (29 kg and 31 kg) showed fatal VF after two 40-second discharges. If an unmodified TASER X26 has the same safety factor as that reported by McDaniel et al.,[4] then we should never have seen VF.

Webster et al.[15] showed that discharges from a standard TASER X26 can cause VF and that the distance of the current emitting dart from the heart is a determining factor. The darts used here were placed with consistent reference to anatomic landmarks but the specific dart-to-heart distances were not measured. However, the approximate dart-to-heart distances (5–10 cm from the superior dart to the right ventricle and twice this from the inferior dart to the right ventricle) greatly exceeded the average distance of 1.5 cm and the maximum distance of 2.4 cm to the right ventricle where VF was reported by Webster et al.[15] To some extent, this may be related to the thinner body wall and smaller thoracic dimensions in our animals (22–46 kg) when compared with those (54–74 kg) used by Webster et al.[15]

In addition to direct electrical disruption of cardiac rhythm, it has been postulated that deaths associated with EID exposure may result from cardiac instability related to EID-induced lactic acidosis.[31,33] Acidosis at pH <7.20 can lower the VF threshold, cause hyperkalemia, and reduce cardiac output.[34] The profound metabolic and respiratory acidosis observed here was caused by the extreme degree of repetitive, global skeletal muscle contraction, by apnea, or by severe circulatory dysfunction.

In this regard, our findings concur with those of Jauchem et al.,[14] where TASER X26 discharges in anesthetized swine caused severe acidosis (pH <7.0) accompanied by dramatic hypercapnia ($P_{CO_2} >100$ mm Hg) and elevated lactate (>15 mmol/L). It is known that when swine are exercised to exhaustion, large increases in lactate (>15 mmol/L) and resultant decreases in bicarbonate are seen.[35] These metabolic changes from exhaustive exercise are countered in conscious swine by hyperventilation and resultant decreases in P_{CO_2}.[35] In the Jauchem et al.[14] study, acidosis may have arisen from inadequate spontaneous respiration and a lack of mechanical ventilation. In the present study, all animals were mechanically ventilated except during the two 40-second actual or sham discharge intervals. Immediately postdischarge, the respiratory rate was adjusted upward to meet the minute ventilation demand of each animal. Despite this intervention, clinically significant respiratory and metabolic acidosis persisted after the two 40-second TASER discharges but not after sham discharges.

The combination of severe hypercapnia and acidosis in the presence of hypotension indicates that circulatory function was affected by TASER X26 discharges. The hypercapnia seen in venous samples is similar to that seen with patients in cardiac arrest.[36] When the heart is not contracting

effectively, the tissues will continue to consume oxygen and produce CO_2. The result is a rise in venous CO_2, accompanied by a drop in venous oxygen saturation.[37] The degree of hypercapnia seen in this study is well beyond that which would be expected in the setting of vigorous muscle contraction alone.[35] The blood gas data observed here suggest that circulatory function was severely affected by the TASER discharge and this is confirmed by the observed decrease in BP. Similar cardiac effects were also seen by Nanthakumar et al.,[16] who showed a loss of BP in swine during TASER discharge measured by aortic manometry.

Two cardiac markers, CK-MB and TnI, were assayed here to assess myocardial injury. There were no elevations in CK-MB. TnI showed small, nonsignificant elevations in both the experimental and control groups. The induction and prolonged anesthesia sessions (2–3 hours) employed on the first day of the experiment may have evoked cardiac stress that contributed to these minor elevations in TnI.[38] Anesthesia, especially at induction, is a known cardiac stressor, which results in an increased risk of adverse cardiac events.

The present study has examined the effects of the TASER X26 using thoracic discharges with a transcardiac vector in anesthetized healthy swine. It does, however, have some limitations. (1) The number of animals used was relatively small but was counter-balanced by the high inter-animal reproducibility of the results. (2) For ethical reasons, ketamine/xylazine anesthesia was used in this swine model. Anesthesia precludes pain perception, which is one of the two principal effects of TASER discharges in conscious humans. Pain perception would undoubtedly alter some of the responses reported here. (3) Only one vector of discharge (transcardiac) was utilized. Alternate discharge vectors may result in greater or lesser myocardial capture.[16] (4) In the field, TASERs are used to subdue combative individuals who are usually in a state of greatly increased sympathetic activity and, in many cases, are under the influence of alcohol or other drugs, which may alter the thresholds for dysrhythmia and for pain. Under those conditions, the effects of TASER discharge might deviate considerably from those seen here.[11] (5) Only two 40-second discharges were used here. These lengthy discharges may have contributed to the incidence of VF, but Webster et al. used 5-second discharges and still observed VF.

The results of this study are in accordance with other published animal studies[14,16] that have used standard, law enforcement-grade TASER X26 devices to study effects in swine. However, they are at variance with those obtained using custom-built TASER-like devices.[4,11] In this swine model, lengthy thoracic discharges from a TASER X26 produced a reversible cardiorespiratory dysfunction which, when coupled with intense muscle contractions, resulted in severe acidosis, tachycardia, hypotension, and sometimes fatal VF. The cardiac capture and VF reported here may be facilitated by the vector of the current, the proximity of the emitting probe to the heart, or the temporal relationship of the discharge pulses to the vulnerable phase of the heart. This model of thoracic TASER discharge indicates that risk of cardiac dysrhythmia exists when the heart is interposed between the darts.

ACKNOWLEDGMENTS

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REFERENCES

1. Bozeman WP. Withdrawal of taser electroshock devices: too much, too soon. *Ann Emerg Med.* 2005;46:300–301.
2. Battershill P, Naughton B, Laur D, Panton K, Massine M, Anthony R. Taser technology review and interim recommendations. Available at: www.cprc.org/docs/bcopcc_final.pdf, 1–59; 2005.
3. Bleetman A, Steyn R. The advanced Taser: a medical review. Available at: www2.warwick.ac.uk/fac/med/healthcom/emergencycare/research, 1–30; 2003.
4. McDaniel WC, Stratbucker RA, Nerheim M, Brewer JE. Cardiac safety of neuromuscular incapacitating defensive devices. *Pacing Clin Electrophysiol.* 2005;28(Suppl 1):S284–S287.
5. Bleetman A, Steyn R, Lee C. Introduction of the Taser into British policing. Implications for UK emergency departments: an overview of electronic weaponry. *Emerg Med J.* 2004;21:136–140.
6. Roy OZ, Podgorski AS. Tests on a shocking device—the stun gun. *Med Biol Eng Comput.* 1989;27:445–448.
7. Ruggieri JA. Forensic engineering analysis of electro-shock weapon safety. *J Natl Acad Forensic Eng.* 2005;22:1–34.
8. Ordog GJ, Wasserberger J, Schlater T, Balasubramanium S. Electronic gun (Taser) injuries. *Ann Emerg Med.* 1987;16:73–78.
9. O'Brien DJ. Electronic weaponry—a question of safety. *Ann Emerg Med.* 1991;20:583–587.
10. Koscove EM. The Taser weapon: a new emergency medicine problem. *Ann Emerg Med.* 1985;14:1205–1208.
11. Lakkireddy D, Wallick D, Ryschon K, et al. Effects of cocaine intoxication on the threshold for stun gun induction of ventricular fibrillation. *J Am Coll Cardiol.* 2006;48:805–811.
12. Ho JD, Miner JR, Lakireddy DR, Bultman LL, Heegaard WG. Cardiovascular and physiologic effects of conducted electrical weapon discharge in resting adults. *Acad Emerg Med.* 2006;13:589–595.
13. Levine SD, Sloane C, Chan T, Vilke T, Dunford J. Cardiac monitoring of subjects exposed to the Taser. *Acad Emerg Med.* 2005;12:71.
14. Jauchem JR, Sherry CJ, Fines DA, Cook MC. Acidosis, lactate, electrolytes, muscle enzymes, and other factors in the blood of Sus scrofa following repeated TASER exposures. *Forensic Sci Int.* 2005;161:20–30.
15. Webster JG, Will JA, Sun H, et al. Can TASERS directly cause ventricular fibrillation. *IFMBE Proc.* 2006;14:3307–3310.
16. Nanthakumar K, Billingsley IM, Masse S, et al. Cardiac electrophysiological consequences of neuromuscular incapacitating device discharges. *J Am Coll Cardiol.* 2006;48:798–804.
17. Lee RC. Injury by electrical forces: pathophysiology, manifestations, and therapy. *Curr Probl Surg.* 1997;34:677–764.
18. Lee RC, Zhang D, Hannig J. Biophysical injury mechanisms in electrical shock trauma. *Annu Rev Biomed Eng.* 2000;2:477–509.
19. Swindle MM. *Surgery, Anesthesia, and Experimental Techniques in Swine.* Ames: Iowa State University Press; 1998.
20. Bollen PJA, Hansen AK, Rasmussen HJ. Important biological features. In: Suckow MA, ed. *The Laboratory Swine.* Boca Raton: CRC Press; 2000:1–14.
21. Maier A, Nance P, Price P, Sherry CJ, Reilly JP, Klauenberg BJ, et al. The Joint Non-Lethal Weapons Human Effects Centre of

Excellence. Human effectiveness and risk characterization of the electromuscular incapacitating device: a limited analysis of TASER: part 11. Appendix A, p. 12. Available at: http://www.taser.com/documents/Part%20II%20NLW%20-%20EMI%20Appendices%20Public%20V1%20Mar%2005%20final.pdf#search=%22The%20Joint%20Non-Lethal%20Weapons%20Human%20Effects%20Centre%20of%20Excellence.%22; 2005.

22. Blackwell V. Chief questions use of Taser on disabled woman. Available at: http://www.firstcoastnews.com/news/news-article.aspx?storyid=75795; 2007.
23. Apple FS, Christenson RH, Valdes R Jr, et al. Simultaneous rapid measurement of whole blood myoglobin, creatine kinase MB, and cardiac troponin I by the triage cardiac panel for detection of myocardial infarction. *Clin Chem.* 1999;45:199–205.
24. Apple FS, Murakami MM, Quist HH, Pearce LA, Wieczorek S, Wu AH. Prognostic value of the Ortho Vitros cardiac troponin I assay in patients with symptoms of myocardial ischemia. Risk stratification using European Society of Cardiology/American College of Cardiology recommended cutoff values. *Am J Clin Pathol.* 2003; 120:114–120.
25. Apple FS, Murakami M, Panteghini M, et al. International survey on the use of cardiac markers. *Clin Chem.* 2001;47:587–588.
26. Ng SM, Krishnaswamy P, Morissey R, Clopton P, Fitzgerald R, Maisel AS. Ninety-minute accelerated critical pathway for chest pain evaluation. *Am J Cardiol.* 2001;88:611–617.
27. Ng SM, Krishnaswamy P, Morrisey R, Clopton P, Fitzgerald R, Maisel AS. Mitigation of the clinical significance of spurious elevations of cardiac troponin I in settings of coronary ischemia using serial testing of multiple cardiac markers. *Am J Cardiol.* 2001; 87:994–999.
28. Perry SV. The regulation of contractile activity in muscle. *Biochem Soc Trans.* 1979;7:593–617.
29. Apple FS, Wu AH. Myocardial infarction redefined: role of cardiac troponin testing. *Clin Chem.* 2001;47:377–379.
30. Schlosberg M, Levin J, Batliwalla S, Daniels J. Stun gun fallacy: how the lack of Taser regulation endangers lives. Available at: http://aclunc.org/police/051006-taser_report.pdf; 2005.
31. Amnesty International. United States of America Excessive and Lethal Force? Amnesty International's concerns about deaths and ill-treatment involving police use of Tasers. Available at: http://www.amnestyusa.org/countries/usa/Taser_report.pdf; 2006.
32. Link MS, Wang PJ, Pandian NG, et al. An experimental model of sudden death due to low-energy chest wall impact. *New Engl J Med.* 1998;338:1805–1811.
33. Stratton SJ, Rogers C, Brickett K, Gruzinski G. Factors associated with sudden death of individuals requiring restraint for excited delirium. *Am J Emerg Med.* 2001;19:187–191.
34. Adrogue HJ, Madias NE. Management of life-threatening acid-base disorders. First of two parts. *N Engl J Med.* 1998;338:26–34.
35. Hastings AB, White FC, Sanders TM, Bloor CM. Comparative physiological responses to exercise stress. *J Appl Physiol.* 1982; 52:1077–1083.
36. Adrogue HJ, Rashad MN, Gorin AB, Yacoub J, Madias NE. Assessing acid-base status in circulatory failure. Differences between arterial and central venous blood. *N Engl J Med.* 1989;320:1312–1316.
37. Martin L. *All You Really Need to Know to Interpret Arterial Blood Gases.* Philadelphia: Lippincott Williams and Wilkins; 1999.
38. Townsend CM, Beauchamp DR, Evers MB, Mattox KL, Sabiston DC. *Sabiston Textbook of Surgery.* Philadelphia: Saunders; 2004:425–426.

Editor's Note: Due to a transcription error, the discussion for this paper by Frederic J. Cole, Jr., MD and others will not appear.

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Research in Brief

Police Use of Force, Tasers and Other Less-Lethal Weapons

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Police Use of Force, Tasers and Other Less-Lethal Weapons

This Research in Brief is based primarily on "A Multi-Method Evaluation of Police Use of Force Outcomes," final report to the National Institute of Justice, July 2010, NCJ 231176, available online at http://www.ncjrs.gov/pdffiles1/nij/grants/231176.pdf.

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ABOUT THIS REPORT

This study looked at injuries that occur to law enforcement officers and citizens during use-of-force events. Most applications of force are minimal, with officers using their hands, arms or bodies to push or pull against a suspect to gain control. Officers are also trained to use various other force techniques and weapons to overcome resistance. These include less-lethal weapons such as pepper spray, batons or conducted energy devices (CEDs) such as Tasers. They can also use firearms to defend themselves or others against threats of death or serious bodily injuries.

What did the researchers find?

This study found that when officers used force, injury rates to citizens ranged from 17 to 64 percent, depending on the agency, while officer injury rates ranged from 10 to 20 percent. Most injuries involve minor bruises, strains and abrasions.

The study's most significant finding is that, while results were not uniform across all agencies, the use of pepper spray and CEDs can significantly reduce injuries to suspects and the use of CEDs can decrease injuries to officers.

The researchers assert that all injuries must be taken seriously. When police in a democracy use force and injury results, concern about police abuse arises, lawsuits often follow and the reputation of the police is threatened. Injuries also cost money in medical bills for indigent suspects, workers' compensation claims for injured officers or damages paid out in legal settlements or judgments.

What were the study's limitations?

In many cases, agency-supplied injury data did not allow for a detailed analysis of the nature or seriousness of the injuries reported.

Geoffrey P. Alpert, Michael R. Smith, Robert J. Kaminski, Lorie A. Fridell, John MacDonald, and Bruce Kubu

Police Use of Force, Tasers and Other Less-Lethal Weapons

Introduction

Police weaponry has come full circle.

During the middle of the 19th century, police officers in New York and Boston relied on less-lethal weapons, mostly wooden clubs. By late in the century, police departments began issuing firearms to officers in response to better armed criminals. Although firearms are still standard issue, law enforcement agencies are again stressing the use of less-lethal weapons rather than firearms.[1]

The Fourth Amendment forbids unreasonable searches and seizures, and various other legal and policy controls govern how and when officers can use force. Most agencies tightly control the use of force and supervisors or internal affairs units routinely review serious incidents. New technologies have added to the concerns about the use of force by law enforcement.

New technologies raise questions

During the past 20 years, new technologies have emerged that offer the promise of more effective control over resistive suspects with fewer or less serious injuries. Pepper spray was among the first of these newer less-lethal weapons to achieve widespread adoption by police forces, and more recently, conducted energy devices (CEDs) such as the Taser have become popular.

Taser use has increased in recent years. More than 15,000 law enforcement and military agencies use them. Tasers have caused controversy (as did pepper spray) and have been associated with in-custody deaths and allegations of overuse and intentional abuse. Organizations such as Amnesty International and the American Civil Liberties Union have questioned whether Tasers can be used safely, and what role their use plays in injuries and in-custody deaths.

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Several studies found that when agencies adopted the use of pepper spray, they subsequently had large declines in assaults on officers and declines in officer and suspect injury rates, and associated injuries were usually minor. Pepper spray provides a way to reduce injuries.

CEDs such as Tasers produce 50,000 volts of electricity. The electricity stuns and temporarily disables people by causing involuntary muscle contractions. This makes people easier to arrest or subdue. When CEDs cause involuntary muscle contractions, the contractions cause people to fall. Some people have experienced serious head injuries or bone breaks from the falls, and at least six deaths have occurred because of head injuries suffered during falls following CED exposure. More than 200 Americans have died after being shocked by Tasers. Some were normal, healthy adults; others were chemically dependent or had heart disease or mental illness.[2]

Tasers use compressed nitrogen to fire two barbed probes (which are sometimes called darts) at suspects. Electricity travels along thin wires attached to the probes. (A new wireless Taser is also on the market.) Darts may cause puncture wounds or burns. A puncture wound to the eye could cause blindness.[3]

Despite the dangers, most CED shocks produce no serious injuries. A study by Wake Forest University researchers found that 99.7 percent of people who were shocked by CEDs suffered no injuries or minor injuries only. A small number suffered significant and potentially lethal injuries.

This NIJ-sponsored study included six police departments and evaluated the results of 962 "real world" CED uses. Skin punctures from CED probes were common, accounting for 83 percent of mild injuries.[4]

Policymakers and law enforcement officials want to know whether Tasers are safe and effective, and how (if at all) they should be used to match police use-of-force choices with levels of suspect resistance. This study indicates that CED use actually decreases the likelihood of suspect injury.

Previous research on use of force and injuries

The controversy around Taser use is not unique. Law enforcement agencies found themselves in similar circumstances with pepper spray in the 1990s. Human rights groups such as Amnesty International questioned the safety and misuse of pepper spray as its use spread rapidly in American law enforcement agencies. NIJ

funded various studies on the safety and effectiveness of pepper spray.[5]

Some studies have focused on officer injury. Several found that about 10 percent of officers were injured when force was used.[6] However, two studies of major police departments found officer injury rates of 38 and 25 percent.[7] The agencies with lower rates allowed officers to use pepper spray, while the two with higher rates did not.

A few researchers have looked at how various approaches to force affect officer injury rates.[8] Overall, the empirical evidence shows that getting close to suspects to use hands-on tactics increases the likelihood of officer injuries. Research also shows that suspects have a higher likelihood of injury when officers use canines, bodily force or impact weapons such as batons. Alternatives to bodily force and impact weapons are found in other less-lethal weapons such as pepper spray and CEDs.

Previous studies on pepper spray and CEDs

Pepper spray. Law enforcement agencies rapidly adopted pepper spray in the late 1980s and early 1990s as an alternative to traditional chemical agents such as tear gas, but its use sparked controversy. Notably, the American Civil Liberties Union of Southern California asserted that pepper spray was causing in-custody deaths. NIJ studies on the link between pepper spray and in-custody deaths found that the deaths were largely a result of positional asphyxia, pre-existing health conditions or were drug related.[9]

Several studies found that when agencies adopted the use of pepper spray, they subsequently had large declines in assaults on officers and declines in officer and suspect injury rates, and associated injuries were usually minor.[10] Pepper spray provides a way to reduce injuries.

CEDs. Many law enforcement agencies noted that injury rates for officers and suspects declined after they introduced CEDs.[11]

Medical research, including controlled animal trials and controlled human trials, has produced various insights. Some animal studies were conducted to learn if CED

use could result in ventricular fibrillation. Several studies showed that standard shocks that lasted five to 15 seconds did not induce ventricular fibrillation of the heart. Higher discharges, 15 to 20 times the standard, or those of longer duration — two 40-second exposures — induced fibrillation or increased heart rhythm in some pigs. In addition, longer exposures led to ventricular fibrillation-induced death in three pigs.[12]

Controlled studies involving healthy human subjects (often law enforcement trainees) found that subjects experienced significant increases in heart rates following exposure, but none experienced ventricular fibrillation.[13]

NIJ study and recommendations

NIJ gathered an expert panel of medical professionals to study in-custody deaths related to CEDs. In its report, the panel said that while CED use is not risk free, there is no clear medical evidence that shows a high risk of serious injury or death from the direct effects of CEDs. Field experience with CED use shows that exposure is usually safe. Therefore, law enforcement agencies need not avoid using CEDs provided they are used in line with accepted national guidelines.[14]

A preliminary review of deaths following CED exposure found that many are associated with continuous or repeated shocks. There may be circumstances in which repeated or continuous exposure is required, but law enforcement officers should be aware that the associated risks are unknown. Therefore, caution is urged in using multiple activations.[15]

The seeming safety margins of CED use on normal healthy adults may not be applicable to small children, those with diseased hearts, the elderly, those who are pregnant and other at-risk people. The use of CEDs against these populations (when recognized) should be avoided, but may be necessary if conditions exclude other reasonable choices.[16]

A suspect's underlying medical conditions may be responsible for behavior that leads law enforcement officers to subdue him or her. Sometimes this includes CED use. Abnormal mental status in a combative or resistive subject, sometimes called

"excited delirium," may be associated with a risk for sudden death. This should be treated as a medical emergency.[17]

The national survey

The Police Executive Research Forum conducted a survey of state, county and municipal law enforcement agencies to learn more about less-lethal technologies and related policies and training. More than 500 agencies participated.

Most agencies have a "use-of-force continuum" that is covered in training, where officers learn to use suitable force levels depending on circumstances. For example, an officer might start by using verbal commands when dealing with a suspect. Then an officer might move to soft empty-hand tactics (such as pushing) when faced with lack of cooperation or mild resistance. The continuum covers various circumstances up to the use of firearms.

The survey included various levels of resistance and asked agencies to describe what force they allow in each. Most agencies allow only soft tactics against a subject who refuses, without physical force, to comply with commands. Just under half allow officers to use chemical weapons at that point. However, if the subject tensed and pulled when an officer tried to handcuff him or her, most agencies would allow chemical agents and hard empty-hand tactics, such as punching. Many also allow for CED use at this point but about 40 percent do not. Almost three-fourths allow CED use if the suspect flees, and almost all allow it when the subject assumes a boxer's stance. Most agencies do not allow baton use until the subject threatens the officer by assuming the boxer's stance.

Three-fourths of the surveyed agencies that use CEDs issued them between 2004 and 2006. Most are using Tasers. In most agencies, officers receive four or six hours of training, and 63.7 percent of agencies require that officers experience activation (i.e., get shocked) during training.

Most agencies do not allow CED use against a subject who nonviolently refuses to comply with commands. However, six in 10 allow for CED use against a subject who tenses and pulls when the officer tries to handcuff him or her. Agencies usually

The seeming safety margins of CED use on normal healthy adults may not be applicable to small children, those with diseased hearts, the elderly, those who are pregnant and other at-risk people. The use of CEDs against these populations (when recognized) should be avoided but may be necessary if conditions exclude other reasonable choices.

place the CED with chemical agents in their force continuum, meaning that their use is typically approved in the same circumstances in which pepper spray use is allowed. CEDs are usually lower on the continuum than impact weapons.

One facet of the controversy surrounding CED use concerns vulnerable populations and circumstances that pose potentially heightened risk to the subject. For only one circumstance — when a subject is near flammable substances — do most agencies (69.6 percent) ban CED use.

Some 31 percent forbid CED use against clearly pregnant women, 25.9 percent against drivers of moving vehicles, 23.3 percent against handcuffed suspects, 23.2 percent against people in elevated areas and 10 percent against the elderly. However, many agencies, while not forbidding use in these circumstances, do restrict CED use except in necessary, special circumstances.

Analysis of information from specific law enforcement agencies

Looking at the experiences of specific agencies can yield important information that might otherwise be lost in larger analyses. The researchers used various statistical techniques to identify factors that increase or decrease the odds of injury to officers and suspects alike.

Richland County Sheriff's Department. The Richland County Sheriff's Department (RCSD) includes about 475 sworn officers who serve the unincorporated portions of Richland County, S.C. Deputies carry Glock .40 caliber pistols, collapsible metal batons and pepper spray. Increasingly, they also carry the model X-26 Taser. The agency started phasing in Taser use in late 2004. During data collection, about 60 percent of deputies carried Tasers.

Researchers coded 467 use-of-force reports covering the period from January 2005 to July 2006. Of the 49 separate injuries recorded for officers (three officers had more than one injury), 46 involved bruises, abrasions or cuts. The department recorded 92

suspect injuries; 69 of those were bruises, abrasions or cuts. Most of the remaining suspect injuries were dog bites, but three involved broken bones or internal injuries.

Further analysis of the data included identifying how various factors increased or decreased the risk of injury to officers or suspects. The use of soft empty-hand techniques by an officer, active aggression by a suspect and suspect use of deadly force all increased the risk for deputies.

Soft empty-hand control was the most frequent force level used by deputies, occurring in 59 percent of all use-of-force incidents. These techniques increased the odds of officer injury by 160 percent. Thus, deputies were at greatest risk for injury when using the least force possible.

Two variables significantly decreased the risk for suspects. Pepper spray use decreased the odds of suspect injury by almost 70 percent, and a deputy aiming a gun at a suspect reduced injury odds by more than 80 percent (because the act of pointing a gun alone often effectively ends the suspect's resistance).

However, the use of a canine posed, by far, the greatest injury risk to suspects, increasing injury odds by almost 40 fold. Suspects who displayed active aggression toward deputies were also more likely to suffer injuries. CED use had no effect on the likelihood of injury; this is inconsistent with the experiences of other agencies, suggesting that not every agency's experience with the Taser will be the same.

Miami-Dade Police Department. The department has about 3,000 officers, is the largest law enforcement agency in the Southeast and is one of the largest departments that has never issued pepper spray to its officers.[18]

The researchers examined 762 use-of-force incidents involving a lone officer and a lone suspect that occurred between January 2002 and May 2006. About 70 percent of the officers carried Tasers by May 2006. Officers were substantially less likely to be injured than suspects, with 16.6 percent (124) of officers injured and 56.3 percent (414) of suspects injured. Most injuries were minor, but 73 suspects (17 percent)

suffered serious injuries. Minor injuries included bruises, sprains and lacerations. Major injuries included bites, punctures, broken bones, internal injuries and gunshot wounds.

The department does not issue pepper spray to its line officers, and there were few incidents involving guns or batons. Analysis of the incidents found that the use of both soft-hand tactics and hard-hand tactics by officers more than doubled the odds of officer injury. Conversely, CED use was associated with a 68-percent reduction in the odds of officer injury.

As for suspects, hands-on tactics increased the odds of injury, the use of canines greatly increased the odds and CED use substantially decreased the odds.

Seattle Police Department. The Seattle Police Department has about 1,200 sworn officers. The agency started using Tasers in December 2000. Other less-lethal weapons include pepper spray, batons and shotgun beanbag rounds. The department recorded 676 use-of-force incidents between Dec. 1, 2005, and Oct. 7, 2006. Suspects suffered injuries in 64 percent of the incidents, while officers suffered injuries in 20 percent of the incidents. Officers used hands-on tactics in 76 percent of the incidents. The next most frequent type of force officers used was the Taser (36 percent), followed by pepper spray (8 percent).

Suspects were impaired by alcohol, drugs or mental illness in 76 percent of the incidents. Just over half (52 percent) of the suspects were nonwhite, and 95 percent were male. Analysis of the data revealed that Taser use was associated with a 48-percent decrease in the odds of suspect injury but did not affect officer injury.

The use of unarmed tactics by officers increased the odds of officer injury 258 percent. The odds of officer injury increased significantly when suspects resisted using physical force or the use or threat of use of a weapon.

Although results were not uniform across the agencies, the analysis shows that the use of pepper spray and CEDs can have a significant and positive injury-reduction effect.

Interestingly, nonwhite suspects were less likely to be injured than whites in both

agencies (Miami and Seattle) where suspects' race was available as a variable for analysis. Another important finding concerns the use of canines. While canines were used rarely, their use substantially increased the risk of injury to suspects in two of the agencies.

Combined agency analysis and its limitations

The researchers also conducted a combined analysis of use-of-force data from 12 large local law enforcement agencies.[19] The full report gives a detailed description of the information available and the limits of the data. Most agencies, for example, had details about demographic characteristics of suspects, but only four had officer demographic information. Moreover, the Miami-Dade Police Department did not use pepper spray while San Antonio did not use CEDs.

Despite the limitations, the study's use of a large sample, representing more than 25,000 use-of-force incidents, allowed the researchers to use statistical techniques in an effort to learn which variables are likely to affect injury rates to officers and suspects. The use of physical force (hands, feet, fists) by officers increased the odds of injury to officers and suspects alike. However, pepper spray and CED use decreased the likelihood of suspect injury by 65 and 70 percent respectively. Officer injuries were unaffected by CED use, while the odds of officer injury increased about 21 percent with pepper spray use.

The researchers noted the 12-agency analysis yielded puzzling results about the relationship between pepper spray use and officer injury rates. Those results are inconsistent with the single agency analysis. More research may explain the differences.

Longitudinal analysis

The researchers reviewed use-of-force information from police departments in Austin, Texas, and Orlando, Fla., to learn how introducing CEDs affected injury rates. This quasi-experimental approach tracked injuries before and after CED introduction.

The Orlando data include 4,222 incidents covering 1998 to 2006. CED use began in February 2003. The Austin data includes 6,596 incidents from 2002 to 2006. However, CED use was

phased in beginning in 2003 and was not completed until June 2004. A large drop in injury rates for suspects and officers alike occurred in both cities following CED introduction.

In both cities, Taser adoption was associated with a statistically significant drop in average monthly injuries to suspects. In Orlando, the suspect injury rate dropped by more than 50 percent compared to the pre-Taser injury rate. In Austin, suspect injury rates were 30 percent lower after full-scale Taser deployment.

In Orlando, the decline in officer injury rates were even greater than for suspects; the average monthly rate dropped by 60 percent after Taser adoption. In Austin, officer injuries dropped by 25 percent.

Interviews with officers and suspects

Researchers conducted interviews with 219 officers from South Carolina's Richland County Sheriff's Department, 35 from the Columbia Police Department (CPD), and 35 suspects involved in use-of-force situations to supplement and add a qualitative context to their quantitative analyses. Generally, they tried to contact officers and suspects within 48 hours of receiving a use-of-force report. Interviews were voluntary, and some officers and suspects declined to participate.

In nine out of 105 use-of-force incidents, Richland County Sheriff's Department officers reported that a Taser did not work properly or did not have the desired effect. In addition, researchers received reports of multiple Taser hits on a suspect and multiple uses of the Taser in "drive stun" mode (when the Taser is pressed against a suspect rather than firing darts) to control suspects (or, based on the suspects' reports, as punishment). These reports indicate that some officers are using Tasers multiple times during an encounter.

Nine percent of the officers reported injuries, almost all of which were scrapes, cuts or bruises suffered while struggling with resistant suspects. Officers also reported that 26 suspects (12 percent) were injured. Most suspect injuries were cuts or abrasions, but there were also two dog bites, and one suspect was shot in the arm after firing at officers.

In 22 cases, researchers interviewed both the officers and suspects involved in an incident. Most suspects said officers used excessive or unnecessary force to subdue them. Some suspects said officers used Tasers quickly, and several said the officers enjoyed watching them endure the pain. Some suspects said officers kneed them in the back and kicked or punched them after they were in handcuffs. Some also said officers used Tasers on them after they were handcuffed.

Suspects often tell a different story than the officers who arrest them. In almost all cases, suspects said officers used excessive force and that they were not resisting arrest. The officers, for their part, said they used minimal force to control suspects, and did not mention using force after a suspect was under control. Officers reported that the force used was necessary and reasonable. In a typical account, a suspect said he was unaware there was a warrant out for his arrest, and when police confronted him, he did not resist. He said the officers "pushed me to the ground and put the cuffs on … they didn't have to do that to me." He said that all the officers had to do was tell him to "quit acting up." He complained that officers should just have told him to calm down instead of pushing him to the ground. By contrast, they said the suspect ran away when confronted, so they tackled him. These kinds of contradictions were common; suspects said they did not resist, and officers provided justification for the force levels they used.

In other cases, suspects and officers offered radically different versions of events. For example, in one case, an officer said he saw several traffic violations and the suspects sped off and stopped, with one suspect running away. The officers said the driver then tried to exit the vehicle from the passenger's side holding a shotgun. One officer pointed his weapon at the suspect, who then dropped the shotgun. The suspect failed to mention the shotgun to researchers and only complained that officers put the handcuffs on too tightly and slammed him around in the back of the transport vehicle.

Unlike the Richland County Sheriff's Department, the

Columbia Police Department did not use Tasers. The officers described 35 use-of-force incidents. Three officers reported that pepper spray was ineffective. In all three cases, the suspects were either drunk or high on drugs. One case, in particular, highlighted the potential advantages of the Taser over pepper spray in some circumstances. In that case, a 6'7", 370-pound man wanted for domestic violence charged an officer with a metal object in his hand. The officer used pepper spray, but it had no effect. The suspect then retreated to the apartment kitchen and grabbed a knife. The officers pointed their guns at him and ordered him to drop the knife, but he refused. He cut and stabbed himself with the knife while the officers waited for another agency to arrive that was equipped with a Taser. The suspect cut himself more than 100 times before the South Carolina Law Enforcement Division arrived and used a Taser on him. The Taser had an instant effect, and officers were then able to handcuff the suspect.

Most injuries in both agencies occurred when officers and suspects struggled on the ground. The differences between the agencies were striking. RCSD equips most of its deputies with Tasers. The deputies collectively reported fewer injuries to themselves and suspects from ground fighting than did CPD officers. CPD did not issue Tasers, and 31 percent of its officers reported getting cuts, scrapes and bruises from wrestling with suspects on the ground. The prevalence of ground fighting injuries among RCSD officers (less than nine percent) was lower, as were injuries to suspects caused by contact with the ground. Some of the injuries could have been prevented had officers used Tasers instead of hands-on tactics.

Implications for policy, training and future research

Because of the controversial nature and widespread use of CEDs, the researchers explored their use in detail and made recommendations, based on the findings, for whether and how CEDs should fit into the range of less-lethal force alternatives available to law enforcement officers.

Factors affecting injuries

Physical force

The findings clearly show the use of physical force and hands-on control increase the risk of injury to officers and suspects. In Richland County, S.C., soft empty-hand control significantly increased the odds of injury to officers, while hard empty-hand tactics increased the risk of injury to suspects. In Miami-Dade, both types of force increased the risk of injury to both officers and suspects. In Seattle, use of force increased injury risk to officers but not to suspects, while the overall analysis (of 12 agencies) showed increased injury risk to suspects and especially to officers associated with physical force. This increased risk was large. When controlling for the use of CEDs and pepper spray in the overall analysis, using force increased the injury odds to officers by more than 300 percent and to suspects by more than 50 percent.

Suspect resistance

Increasing levels of suspect resistance were associated with an increased risk of injury to officers and suspects. The increased injury risk was especially acute for officers. In Richland County, active aggression and threats of deadly force increased the odds of officer injury by more than 100 percent. The odds of suspect injury were unchanged in Seattle with increased resistance levels. These findings suggest that officers, rather than suspects, face the most increased injury risk when suspects resist more vigorously.

Pepper spray

The findings suggest that, at least for suspects, pepper spray use reduces the likelihood of injury. In Richland County, pepper spray use reduced the odds of suspect injury by 70 percent but did not affect officer injuries. In Seattle, pepper spray use had no effect on injury rates for officers or suspects. However, the overall analysis (of 12 agencies) showed that pepper spray use reduced the likelihood of injury to suspects by 70 percent, which was even more than the decline noted with CEDs (see below). For officers, pepper spray use increased the likelihood of injury by 21 to 39 percent. This finding

was unexpected, and more research may help to explain how officers choose to use pepper spray versus CEDs.

CEDs

Except for in Richland County where its effects were insignificant, CED use substantially decreased the likelihood of suspect injury. In Miami-Dade, the odds of a suspect being injured were almost 90 percent lower when a CED was used than when it was not. Similarly, the odds of suspect injury went down by almost 50 percent when CEDs were used in Seattle. The larger analysis of 12 agencies and more than 24,000 use-of-force cases showed the odds of suspect injury decreased by almost 60 percent when a CED was used. In Richland County, Seattle, and in the larger analysis, Taser use had no effect on officer injuries, while in Miami-Dade, officer injuries were less likely when a Taser was used. Controlling for other types of force and resistance, CED use significantly reduced the likelihood of injuries. CED adoption by the Orlando and Austin police departments reduced injuries to suspects and officers over time.

Demographic characteristics

Apart from officer force and suspect resistance, few other factors influenced injury outcomes. In Miami-Dade, male suspects were twice as likely to be injured as females. The same held true for the 12-agency analysis. In that larger analysis, the presence of a male suspect slightly increased injury risk to officers. In Seattle, female officers were more than twice as likely to be injured as male officers.

Placement of pepper spray and CEDs on a linear use-of-force continuum

People rarely die after being pepper sprayed or shocked with a Taser. However, if injury reduction is the primary goal, agencies that allow use of these less-lethal weapons are clearly at an advantage. Both weapons prevent or minimize the physical struggles that are likely to injure officers and suspects alike. Although both cause pain, they reduce injuries, and according to current medical research, death or serious harm associated with their

use is rare. In that sense, both are safe and similarly effective at reducing injuries. Both should be allowed as possible responses to defensive or higher levels of suspect resistance. This recommendation is supported by the findings and is now followed by most agencies that responded to the national survey.

Policy and training issues related to CEDs

CEDs were used far more often (four to five times more often) than pepper spray among agencies that equipped officers with CEDs and were sometimes used at rates that exceeded empty-hand control. Unlike pepper spray, CEDs do not require decontamination and do not carry the risk of accidental "blow back" that often occurs with pepper spray use. However, they do entail the removal of prongs and the potential for an unintended shock to an officer. Even with these concerns, they are rapidly overtaking other force alternatives. Although the injury findings suggest that substituting CEDs for physical control tactics may be useful, their ease of use and popularity among officers raise the specter of overuse.

The possible overuse of CEDs has several dimensions. CEDs can be used inappropriately at low levels of suspect resistance. Law enforcement executives can manage this problem with policies, training, monitoring and accountability systems that provide clear guidance (and consequences) to officers regarding when and under what circumstances CEDs should be used, or when they should not be used.

Besides setting the resistance threshold appropriately, good policies and training would require that officers evaluate the age, size, gender, apparent physical capabilities and health concerns of a suspect. In addition, policies and training should prohibit CED use in the presence of flammable liquids or in circumstances where falling would pose unreasonable risks to the suspect (in elevated areas, adjacent to traffic, etc.). Policies and training should address the use of CEDs on suspects who are controlled (e.g., handcuffed or otherwise restrained) and should either prohibit such use outright or limit them to clearly defined, aggravated circumstances.

In addition to being used too often, CEDs can be used too much. Deaths associated with CED use often involve multiple Taser activations (more than one Taser at a time) or multiple five-second cycles from a single Taser. CED policies should require officers to assess continued resistance after each standard cycle and should limit use to no more than three standard cycles. Following CED deployment, the suspect should be carefully observed for signs of distress and should be medically evaluated at the earliest opportunity.

Directions for future research

CEDs can be used too much and too often. A critical research question focuses on the possibility of officers becoming too reliant on CEDs. During interviews with officers and trainers, the researchers heard comments that hinted at a "lazy cop" syndrome. Some officers may turn to a CED too early in an encounter and may relying on a CED rather than rely on the officer's conflict resolution skills or even necessary hands-on applications. Research should explore how officers who have CEDs perceive threats, compared to officers who do not have them. In addition, it is important to determine when, during an encounter, an officer deploys the CED.

Another important CED-related research project would be a case study of in-custody deaths involving CED use and a matched sample of in-custody deaths when no CED use occurred. Advocacy groups argue that CEDs can cause or contribute to suspect deaths. The subjects in CED experimental settings have all been healthy people in relatively good physical condition who are not under the influence of alcohol or drugs. There is no ethical way to expose overweight suspects who have been fighting or using drugs to the effects of CEDs, so an examination of cases where similar subjects lived and died may shed some light on the reasons for the deaths. Law enforcement officials typically argue that most if not all the subjects who died when shocked by a CED would have died if the officers had controlled and arrested them in a more traditional hands-on fight. At this point, the argument is rhetorical and research is needed to understand the differences and similarities in cases where suspects died

in police custody, including deaths where a CED may or may not have been involved.

Finally, female officers in Seattle were more than twice as likely to suffer injuries as males. Perhaps the finding in Seattle is an anomaly, but it should be investigated further.

Notes

1. Police Executive Research Forum, "Comparing Safety Outcomes in Police Use-of-Force Cases for Law Enforcement Agencies That Have Deployed Conducted Energy Devices and a Matched Comparison Group That Have Not: A Quasi-Experimental Evaluation," report submitted to the National Institute of Justice, grant number 2006-IJ-CX-0028, 2009: 13.

2. http://www.ojp.usdoj.gov/nij/topics/technology/less-lethal/how-ceds-work.htm.

3. Ibid.

4. http://www.ojp.usdoj.gov/nij/topics/technology/less-lethal/monitoring-ced-use.htm.

5. Edwards, S.M., J. Granfield, and J. Onnen, *Evaluation of Pepper Spray,* Research in Brief, Washington, D.C.: U.S. Department of Justice, National Institute of Justice, February 1997, NCJ 162358; Granfield, J., J. Onnen, and C.S. Petty, *Pepper Spray and In-Custody Deaths,* Alexandria, Va.: International Association of Chiefs of Police, 1994; Petty, C.S., "Deaths in Police Confrontations When Oleoresin Capsicum Is Used," final report, Washington, D.C.: U.S. Department of Justice, National Institute of Justice, 2004, NCJ 204029.

6. Henriquez, M., "IACP National Database Project on Police Use of Force," in *Use of Force by Police: Overview of National and Local Data,* Washington, D.C.: U.S. Department of Justice, National Institute of Justice and Bureau of Justice Statistics, 1999: 19-24; Kaminski, R., C. DiGiovanni, and R. Downs, "The Use of Force Between the Police and Persons With Impaired Judgment," *Police Quarterly,* 7 (2004): 311-338; Smith, M.R., and M. Petrocelli, "The Effectiveness of Force Used by Police in Making Arrests," *Police Practice and Research,* 3 (2002): 201-215.

7. Alpert, G.P., and R.G. Dunham, "Analysis of Police Use-of-Force Data," final report, Washington, D.C.: U.S. Department of Justice, National Institute of Justice, 2000, NCJ 183648; Alpert, G.P., and R.G. Dunham, *Understanding Police Use of Force: Officers, Suspects, and Reciprocity,* Cambridge, NY: Cambridge University Press, 2004; Kaminski, R.J., and D.W.M. Sorensen, "A Multivariate Analysis of Individual, Situational, and Environmental Factors Associated with Police Assault Injuries," *American Journal of Police,* 14 (3/4) (1995): 3-48.

8. See, e.g., Alpert and Dunham, 2000, note 7.

9. Granfield, Onnen, and Petty, 1994, note 5; Petty, 2004, note 5.

10. Edwards, Granfield, and Onnen, 1997, note 5; Kaminski, R.J., S.M. Edwards, and J.W. Johnson, "Assessing the Incapacitative Effects of Pepper Spray During Resistive Encounters With the Police,"

Policing: An International Journal of Police Strategies and Management, 22 (1999): 7-29; Lumb, R.C., and P.C. Friday, "Impact of Pepper Spray Availability on Police Officer Use-of-Force Decisions," *Policing: An International Journal of Police Strategies and Management,* 20 (1997): 136-148; National Institute of Justice, *The Effectiveness and Safety of Pepper Spray,* Research for Practice, Washington, D.C.: U.S. Department of Justice, National Institute of Justice, 2003, NCJ 195739; Nowicki, E., "Oleoresin Capsicum: A Non-Lethal Force Alternative," *Law Enforcement Technology,* 20 (1993): 24-27; Smith and Petrocelli, 2002, note 6.

11. Charlotte-Mecklenburg Police Department, *Taser Project: First Year—Full Deployment Study.* Charlotte, N.C.: Charlotte-Mecklenburg Police Department, 2006; Hougland, S., C. Mesloh, and M. Henych, "Use of Force, Civil Litigation, and the Taser," *FBI Law Enforcement Bulletin,* 74 (2005): 24-30; Jenkinson, E., C. Neeson, and A. Bleetman, "The Relative Risk of Police Use-of-Force Options: Evaluating the Potential for Deployment of Electronic Weaponry," *Journal of Clinical Forensic Medicine,* 13 (2005): 229-241.

12. Dennis, A.J., D.J. Valentino, R.J. Walter, K.K. Nagy, J. Winners, F. Bokhari, D.E. Wiley, K.T. Joseph, and R.R. Roberts, "Acute Effects of TASER X26 Discharges in a Swine Model," *The Journal of Trauma, Injury, Infection and Critical Care,* 63 (2007): 581-590; Esquivel, A., E. Dawe, J. Sala-Mercado, R. Hammond, and C. Bir, "The Physiological Effects of a Conducted Electrical Weapon in Swine," *Annals of Emergency Medicine,* 50 (2007): 576-583; Ho, J.D., J.R. Miner, D.R. Lakireddy, L.L. Bultman, and W.G. Heegaard, "Cardiovascular and Physiologic Effects of Conducted Electrical Weapon Discharge in Resting Adults," *Academic Emergency Medicine,* 13 (2006): 589-595; Lakkireddy, D., D. Wallick, A. Verma, K. Ryschon, W. Kowalewski, O. Wazni, J. Butany, D. Martin, and P.J. Tchou, "Cardiac Effects of Electrical Stun Guns: Does Position of Barbs Contact Make a Difference?" *Pacing and Clinical Electrophysiology,* 31 (2008): 398-408; McDaniel, W.C., R.A. Stratbucker, M. Nerheim, and J.E. Brewer, "Cardiac Safety of Neuromuscular Incapacitating Defensive Devices," *Pacing and Clinical Electrophysiology,* 28 (2005): s284-s287; Nanthakumar, K., I.M. Billingsley, S. Masse, P. Dorian, D. Cameron, V.S. Chauhan, E. Downar, and E. Sevaptsidis, "Cardiac Electrophysiological Consequences of Neuromuscular Incapacitating Device Discharges," *Journal of the American College of Cardiology,* 48 (2006): 798-804; Roy, O.Z., and A.S. Podgorski, "Tests on a Shocking Device — The Stun Gun," *Medical and Biological Engineering and Computing,* 27 (1989): 445-448; Stratbucker, R., R. Roeder, and M. Nerheim, "Cardiac Safety of High Voltage Taser X26 Waveform," Engineering in Medicine and Biology Society, Proceedings of the 25th Annual International Conference of the IEEE EMBS, Cancun, Mexico, 1094-678X, 4 (2003): 3261-3262; Walter, R., A. Dennis, D. Valentina, B. Margeta, K. Nagy, F. Bokhari, D. Wiley, K. Joseph, and R. Roberts, "TASER X26 Discharges in Swine Produce Potentially Fatal Ventricular Arrhythmias," *Academic Emergency Medicine,* 15 (2008): 66-73.

13. Dawes, D.M., J.D. Ho, M.A. Johnson, E. Lundin, T.A. Janchar, and J.R. Miner, "15-Second Conducted Electrical Weapon Exposure

Does Not Cause Core Body Temperature Elevation in Non-Environmentally Stressed Resting Adults," *Forensic Science International,* 176 (2008): 253-257; Dawes, D.M., J.D. Ho, and J.R. Miner, "The Effect of a Cross-Chest Electronic Control Device Exposure on Breathing," *Annals of Emergency Medicine,* 54 (2008): 65; Dawes, D.M., J.D. Ho, M.A. Johnson, E. Lundin, and J.R. Miner, "15-second Conducted Electrical Weapon Application Does Not Impair Basic Respiratory Parameters, Venous Blood Gases, or Blood Chemistries and Does Not Increase Core Body Temperature," *Annals of Emergency Medicine,* 50 (2007): 6; Dawes, D.M., J.D. Ho, M.A. Johnson, E. Lundin, and J.R. Miner, "Breathing Parameters, Venous Blood Gases, and Serum Chemistries With Exposure to a New Wireless Projectile Conducted Electrical Weapon in Human Volunteers," *Annals of Emergency Medicine,* 50 (2007): 133; Ho, J.D., D.M. Dawes, L.L. Bultman, J.L. Thacker, L.D. Skinner, J.M. Bahr, M.A. Johnson, and J.R. Miner, "Respiratory Effect of Prolonged Electrical Weapon Application on Human Volunteers," *Academic Emergency Medicine* 14 (3) (2007): 197-201; Ho, J.D., J.R. Miner, D.R. Lakireddy, L.L. Bultman, and W.G. Heegaard, "Cardiovascular and Physiologic Effects of Conducted Electrical Weapon Discharge in Resting Adults," *Academic Emergency Medicine,* 13 (2007): 589-595; Ho, J.D., D.M. Dawes, R.F. Reardon, A.L. Lapine, and J.R. Miner, "Echocardiographic Determination of Cardiac Rhythm During Trans-Thoracic Wireless Conducted Electrical Weapon Exposure," *Annals of Emergency Medicine,* 52 (2008): 62; Levine, S.D., C. Sloane, T.C. Chan, J. Dunford, and G. Vilke, "Cardiac Monitoring of Human Subjects Exposed to the Taser," *Journal of Emergency Medicine,* 13 (2007): 47; Levine, S.D., C. Sloane, T.C. Chan, G. Vilke, and J. Dunford, "Cardiac Monitoring of Subjects Exposed to the Taser," *Academic Emergency Medicine,* 12 (2005): 71; Vilke, G.M., C. Sloane, K.D. Bouton, F.W. Kolkhorst, S. Levine, T. Neuman, E. Castillo, and T.C. Chan, "Physiological Effects of a Conducted Electrical Weapon on Human Subjects," *Annals of Emergency Medicine,* 26 (2007): 1-4.

14. National Institute of Justice, *Study of Deaths Following Electro Muscular Disruption,* Special Report, Washington, D.C.: U.S. Department of Justice, National Institute of Justice, 2011: 3, NCJ 233432.

15. Ibid., 4.

16. Ibid.

17. Ibid., 5.

18. The MDPD provides police services to the unincorporated areas of Miami-Dade County, Fla., which together contain more than 1 million people in a 1,840 square mile area.

19. The agencies included police and sheriff's departments in Austin, Texas; Cincinnati, Ohio; Harris County, Texas; Hillsborough County, Fla.; Los Angeles (both the city and the county); Miami-Dade, Fla.; Nashville, Tenn.; Orlando, Fla.; Richland County, S.C.; San Antonio, Texas; and Seattle, Wash.

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Sudden Cardiac Arrest and Death Following Application of Shocks From a TASER Electronic Control Device

Douglas P. Zipes, MD

Background—The safety of electronic control devices (ECDs) has been questioned. The goal of this study was to analyze in detail cases of loss of consciousness associated with ECD deployment.

Methods and Results—Eight cases of TASER X26 ECD–induced loss of consciousness were studied. In each instance, when available, police, medical, and emergency response records, ECD dataport interrogation, automated external defibrillator information, ECG strips, depositions, and autopsy results were analyzed. First recorded rhythms were ventricular tachycardia/fibrillation in 6 cases and asystole (after ≈30 minutes of nonresponsiveness) in 1 case. An external defibrillator reported a shockable rhythm in 1 case, but no recording was made. This report offers evidence detailing the mechanism by which an ECD can produce transthoracic stimulation resulting in cardiac electrical capture and ventricular arrhythmias leading to cardiac arrest.

Conclusions—ECD stimulation can cause cardiac electrical capture and provoke cardiac arrest resulting from ventricular tachycardia/ventricular fibrillation. After prolonged ventricular tachycardia/ventricular fibrillation without resuscitation, asystole develops. **(*Circulation*. 2012;125:2417-2422.)**

Key Words: conducted energy weapon injuries ■ death, sudden ■ heart arrest ■ ventricular fibrillation

Electronic control devices (ECDs), also referred to as conducted electrical weapons, are called less lethal or nonlethal weapons because their intent is to incapacitate temporarily, not to kill. The ECD manufactured by TASER International, model X26, is the device most widely used by law enforcement, corrections, and military establishments worldwide. It is a handgun-shaped weapon that uses compressed nitrogen to shoot two 9- or 12-mm barbs from a cartridge into the clothes/skin of an individual. Wires connect the barbs through the cartridge to the gun through which is delivered an initial 50 000-V shock, followed by 100-microsecond pulses at ≈19 Hz (≈1140 times per minute), 2 to 4 amps, 100-microcoulomb charge per pulse, and ≈1200 V.[1] The standard 5-second shock cycle can be halted earlier, repeated, or sustained longer by the user. If electrical currents from both darts connect with the subject, pain and skeletal muscle contraction generally result in rapid incapacitation. The exposed electrodes of the gun can be pressed against the skin (drive stun mode) to cause pain without muscular contractions. ECDs are not considered firearms and therefore are not regulated by the Bureau of Alcohol, Tobacco, Firearms, and Explosives.

Editorial see p 2406
Clinical Perspective on p 2422

The safety of ECDs has been questioned. Amnesty International documented 334 deaths that occurred after exposure to ECDs between 2001 and 2008.[2] To date, no peer-reviewed publication has definitely concluded that ECD shocks can precipitate ventricular fibrillation (VF) causing sudden cardiac arrest and death in humans.

The purpose of this report is to present an analysis of sudden cardiac arrests and deaths following ECD exposure that occurred in 2006 (case 1), in 2008 (cases 2, 4, 5, and 8), and 2009 (cases 3, 6, and 7) and to weigh the existing animal and clinical data to determine whether an ECD shock can cause cardiac electrical capture and, in so doing, provoke ventricular tachycardia (VT) and/or VF in humans.

Methods

The medical information used for this article was approved by the Institutional Review Board of the Indiana University School of Medicine. Written and informed consent was obtained from each person or an authorized representative.

The cases included have been studied as part of litigation related to administration of ECD shocks from the TASER X26 device. In each instance, when available, police, medical, and emergency response records, X26 dataport interrogation, automated external defibrillator information, ECG strips, depositions, and autopsy results were analyzed.

Results

All individuals were previously clinically healthy males who received shocks from the TASER X26 ECD with 1 or both

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Table. Summary of the 8 Cases

Case	Age, y	Height, Weight, lb	ECD Shock(s), s	Response to ECD Shock	Time to Initial ECG After ECD Shock, min	Initial Recorded Rhythm	Drug Screen	Cardiac Findings at Autopsy	Comments
1	48	6 ft 0 in, 155	5, 8, 5	LOC toward end of last ECD cycle	Several	VT/VF	BAC 0.35 g/100 mL; THC present	Survived with memory impairment; normal echocardiogram	Five AED shocks, intravenous epinephrine, and lidocaine eventually restored a perfusing rhythm
2	17	5 ft 7 in, 170	37, 5	LOC toward end of 37-s cycle	>4.5	VF	Negative	410 g; focal atherosclerosis; plaintiff pathologist: normal; defense pathologist: HCM	3 defibrillating shocks and an additional 3 shocks from a second AED at least 9 min after the collapse failed to resuscitate
3	17	5 ft 8 in, 115	5	ILOC	>5	VF	BAC 0.25 g/100 mL; THC present	270 g; normal heart	Asystole developed after the AED shock and then PEA; subsequently, VF recurred and a second AED shock was delivered, followed by asystole/PEA; could not be resuscitated
4	24	5 ft 10 in, 176	11	ILOC	≈10	AED: "shockable rhythm"; asystole after shock; no recordings available	BAC 0.319 g/100 mL	400 g; plaintiff pathologist: no specific pathology; defense pathologist: lymphocytic myocarditis	Said to be breathing initially with a weak radial pulse; resuscitated in hospital; life support withdrawn after 3 d because of anoxic encephalopathy
5	33	6 ft 2 in, 220	13 shocks totaling 62 s in <3 min	LOC toward the end of multiple shocks	≈13	Fine VF vs asystole	Gabapentin 31 μg/mL	470 g; 10%–20% narrowing of the LAD; normal histology	Gabapentin taken for seizure disorder
6	24	5 ft 6 in, 144	49, 5	LOC toward end of 49-s shock	≈10	VT/VF	Negative	366.7 g; normal gross and microscopic findings	Said to be breathing initially; could not be resuscitated
7	16	5 ft 3 in, 130	5	ILOC	≈10	VT/VF	THC	380 g; medical examiner diagnosis: right ventricular cardiomyopathy, disputed by plaintiff's expert	Six AED shocks for VT/VF resulted in asystole/PEA; could not be resuscitated.
8	23	5 ft 9 in, 173	21, 7, 3	LOC toward end of 21-s shock	≈30	Asystole	BAC 0.111 g/100 mL	400 g; mild interstitial fibrosis of compact atrioventricular node; interstitial fibrosis, atrophy, and vacuolization of penetrating and branching bundle	Said to be breathing with pulse initially; could not be resuscitated; cardiac pathologist could not determine whether changes contributed to death

ECD indicates electronic control device; LOC, loss of consciousness during/after initial shock; VT, ventricular tachycardia; VF, ventricular fibrillation; BAC, blood alcohol concentration; THC, tetrahydrocannabinol, positive screen for marijuana; AED, automated external defibrillator; HCM, hypertrophic cardiomyopathy; PEA, pulseless electrical activity; ILOC, immediate loss of consciousness during/after initial shock; and LAD, left anterior descending coronary artery. Heart weight is given in grams. Gabapentin is Neurontin.

barbs in the anterior chest near or over the heart and developed loss of consciousness during or immediately after the ECD shock (Table). First recorded rhythms were VT/VF in 6 and asystole (after ≈30 minutes of nonresponsiveness) in 1 (Figure). An external defibrillator reported a shockable rhythm in 1, but no recording was made. Except for case 1, all died.

Discussion

New Observations

This report adds detailed observations on 8 new cases of sudden cardiac arrest/death following ECD shocks to those already in the literature.

Published Reports

The first published report of sudden cardiac arrest after ECD discharge was a letter to the editor about a 14-year-old boy who immediately lost consciousness after a 17-second ECD chest shock.[3] He initially had a pulse and was breathing but 2 minutes after collapse had VF documented in an ECG recorded by paramedics; he was ultimately resuscitated. The accuracy of statements made in this publication was contested,[4] but sworn testimony by a paramedic who witnessed the entire event[5] stated that VF was recorded 2 minutes after the ECD shock.

The second observation was of a 17-year-old man who received ECD applications of 25 and 5 seconds in the anterior

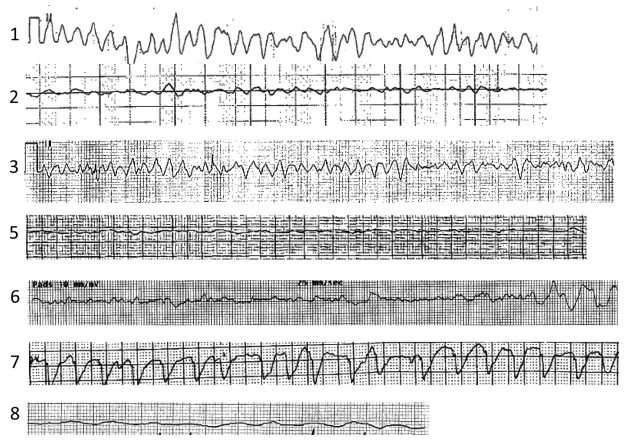

Figure. First available ECG for 7 of the 8 cases.

chest. He immediately dropped to the ground and was observed to become cyanotic and apneic. The initial rhythm was asystole recorded >10 minutes after the ECD application. He was eventually resuscitated with hypothermia[6] but had memory impairment.

The third publication reviewed the presenting rhythm in 56 sudden deaths temporally proximate to discharge of a TASER ECD, finding VF in 4, but concluding that only 1 could be related to ECD discharge. That individual was a 25-year-old man who received ECD shocks in the anterior chest for 16, 5, and 5 seconds. He immediately lost consciousness, and prompt application of an automated external defibrillator showed VF. After 2 shocks, he could not be resuscitated. The report stated that "…the time course and the electrode location are consistent with electrically induced VF."[7]

Animal and Clinical Studies

The concept of cardiac capture by transthoracic electrical impulses in humans was pioneered by Zoll,[8] replicated by many others subsequently,[9] and is now a standard part of resuscitative equipment. The threshold for transthoracic cardiac electrical capture is 100 microcoulombs,[10] which is the output of the TASER model X26.[1]

Studies in pigs,[11–15] sheep,[16] and humans[17] established that transthoracic shocks from the TASER model X26 or a new prototype ECD[18] caused cardiac electrical capture. In addition, porcine research showed that such electrical capture could provoke VF at normal[12–15] or higher-than-normal[11] TASER model X26 outputs. Vectors encompassing the heart and probes closer to the heart facilitated electrical capture.[11,12,15] In 1 study, standard 9-mm probes produced VF in pigs at dart-to-heart distances of 4 to 8 mm.[19] In another porcine study testing barbs at 5 locations, the authors found that 5-second shocks from the TASER X26 produced cardiac electrical capture without VF at normal outputs but that VF induction at increased output was initiated when the capture ratio was ≤2:1. Bracketing the heart with darts in a sternal notch to cardiac apex position along the cardiac axis resulted in the lowest safety margin for VF induction. The authors stated that cardiac disease could reduce the VF threshold and provide a substrate for arrhythmia induction and that rapid ventricular capture was the likely mechanism of VF induction.[20]

In addition to the importance of probe location, longer-duration shocks at normal TASER X26 outputs appear more likely to induce VF.[13,14] Although it is possible that body size might influence cardiac capture and development of VF, clearly big people can still develop VF from an ECD shock (see the Table).

Determining whether an ECD shock caused cardiac electrical capture can be difficult because the shock produces electrical interference in the ECG recording. In a clinical study of a new TASER ECD prototype, cardiac electrical capture monitored echocardiographically was shown to occur at 240 bpm in 1 volunteer.[18] In addition, a case report about a man with an implanted pacemaker demonstrated cardiac electrical capture at rates exceeding 200 bpm during each of two 5-second TASER model X26 applications that was found when the pacemaker was interrogated.[17]

Multiple clinical studies have not shown ECD-induced VF in healthy volunteers. But, because of ethical considerations, even those few studies testing actual barbs to the anterior chest and single 15-second exposures[21] may not be able to replicate the clinical scenario of a frightened/fleeing/fighting individual.

Several epidemiological studies have not shown a link between ECD shocks and sudden cardiac death.[22,23] However, a recent review[24] determined that single shocks in healthy people "…could have deleterious effects when used in the field, in particular if persons receive multiple exposures…or present with medical comorbidities."

Proposed Mechanism of ECD-Induced Sudden Cardiac Arrest

Electrical stimulation can induce VF by causing ventricular capture during the vulnerable period of the T wave of the previous beat or ventricular capture at rates too fast for ventricular activation to remain organized. Rapid pacing also can cause a precipitous blood pressure fall, leading to ischemia. VF by rapid pacing was often the outcome of runaway pacemakers many years ago.[25]

It is clear from the information cited above that an ECD shock to the chest can produce cardiac electrical capture at rapid rates in animals and humans.[11–18] Furthermore, it is clear that VF has been documented as early as 2 minutes after an ECD shock to humans.[3,5] What is lacking is the actual ECG recording of VF induction during an ECD shock in humans, a practical impossibility unless it fortuitously occurred in an individual with a recording device already in place. Even then, electrical interference may obscure the recording. However, ECD-induced VT and VF have been clearly and repeatedly shown in pigs.[11–15,19] In 1 example, intravenous epinephrine in an anesthetized pig, infused at a concentration that increased the spontaneous sinus rate 50% to replicate the clinical "fight or flight" situation, improved the TASER model X26 electrical capture ratio from 3:1 to 2:1 and resulted in VF induction.[12]

Thus, from the pig studies, a likely clinical scenario is that ECD induced cardiac electrical capture at rates of 200 to 240 bpm (a 6:1–4:1 ratio), as already shown in humans.[17,18] The

increased rate plus sympathetic effects can shorten ventricular refractoriness to permit further ECD-induced rate acceleration that eventually causes VF from the rapid rate or R on T. Because a sharp blood pressure reduction results from the rapid rate, repeated shocks or those exceeding the recommended 5-second ECD discharge can add an ischemic component and would be more likely to provoke VF. Furthermore, on the basis of clinical electrophysiological studies performed over many years, the presence of underlying heart disease or arrhythmogenic drugs would be expected to facilitate VF induction by electrical stimulation. Finally, an individual falling forward and then lying prone may be at even greater risk for cardiac capture since the heart can move closer to the chest wall and hence, be closer to the barbs and site of stimulation. The fall might even push the barbs into the skin to a greater depth.

Certainly not every in-custody death occurring after ECD deployment is due to the effects of the ECD shock. Restraint asphyxia and the concept of "excited delirium" are among other explanations.[26] Excited delirium may be a form of takotsubo cardiomyopathy.[27] Considering the fact that extreme sympathetic stimulation likely accompanies most restraint attempts, particularly those with ECD discharge, and the relatively few reported sudden deaths, it seems more logical to conclude that the ECD rather than sympathetic stimulation was responsible for the sudden death.

Alternative explanations such as excited delirium would be more relevant when there was a significant time delay between ECD deployment and loss of consciousness/responsiveness or death.[28] However, when loss of consciousness/responsiveness occurs during/immediately after an ECD chest shot, as it did in each of the cases above, and the subsequent rhythm is VT/VF or asystole (if a long time has elapsed without resuscitation) with no other cause apparent, it becomes difficult to exonerate the effects of the shock. It is also possible that combinations exist. For example, prolonged QT interval in takotsubo cardiomyopathy or metabolic changes from prolonged or repeated shocks might predispose to pacing-induced VT/VF.

Several victims were alleged to have structural heart disease (cases 2, 4, 7, and 8) and/or had elevated blood alcohol concentrations (cases 1, 3, 4, and 8). Although sudden death caused by underlying heart disease or alcohol is possible, one would have to postulate that the heart disease or alcohol coincidentally induced sudden loss of consciousness precisely at the time of ECD application. Far more likely is that stimulation from the ECD in the presence of structural heart disease and/or alcohol intoxication induced VT/VF. Clinical electrophysiology studies over many years have established that the presence of structural heart disease facilitates electrical induction of VT/VF, as does alcohol.[29]

Institutional Reviews

A contemporary review by the National Institute of Justice concluded that the case reported as a letter to the editor[3] was ECD-induced VF and stated that an ECD "may induce rapid ventricular pacing or VT in an individual who appears to be in satisfactory condition…lead[ing] to VF after a short delay," and therefore "use involving the area of the chest in front of the heart area is not totally risk free."[30] The Report of the Braidwood Commission of Inquiry,[31] after an ECD-related death recorded on video in the Vancouver, BC, airport, stated, "There is evidence that the electrical current from a conducted energy weapon is capable of triggering ventricular capture…and that the risk of ventricular fibrillation increases as the tips of the probes get closer to the wall of the heart…[I]f a person dies suddenly and from no obvious cause after being subjected to a conducted energy weapon, death is almost certainly due to an arrhythmia."

Incidence

Sudden death occurs infrequently after ECD deployment, considering the number of ECD applications and the apparently few reported sudden deaths. However, the actual incidence of death when the darts are impaled in the chest is unknown because accurate numerators and denominators are uncertain owing to potential underreporting of total number of sudden deaths (numerator) and the actual number of chest shocks that might cause cardiac electrical capture (denominator). ECD applications without 1 or both probes in the anterior chest would not be expected to influence cardiac rhythm and likely make up a large number of the total applications (denominator) cited. Until a detailed database of ECD deployments and outcomes is created, the exact incidence will remain unknown.

Clinical Implications

It is important to stress that the purpose of this article is not to condemn ECD use by trained professionals. Law enforcement experts must make those decisions, not physicians. Intuitively, one would expect a less lethal weapon to reduce in-custody–related sudden deaths and to be preferable to firearms. Such may not always be the case, however. One study[32] noted that the rate of in-custody sudden deaths increased >6-fold and the rate of firearm deaths increased >2-fold in the first full year after ECD deployment compared with the average rate in the 5 years before deployment.

The main purpose of this article is to make ECD users aware that cardiac arrest caused by VF can result from an ECD shock. Users should be judicious in how and when to use the ECD weapon, avoid chest shocks if possible, as TASER International recommended in September 2009, monitor the person after an ECD shock, and suspect this adverse response in any victim who loses consciousness. Users should be prepared to resuscitate, including deployment of an automated external defibrillator if indicated.

Limitations

The incidence of ECD-induced sudden cardiac arrest/death cannot be determined without accurate data compiled in a national registry of ECD deployments and outcomes. Such a registry should also chart precise dart locations and should be administered and reviewed by an independent oversight group.

The major limitation of this study is not having an ECG recording during ECD application, a practical impossibility in the field situation, as noted earlier. The hard facts of each encounter are 2 events separated in time: ECD deployment and the recorded ECG. The explanation of the cause of the

events in between, ie, the loss of consciousness/responsiveness and sudden death, is based on the animal and clinical data detailed above. However, in cases 4, 6, and 8, reports stated that a pulse and/or respirations were recorded initially, which would seem to be incompatible with ECD-induced VF. Yet, the victims were totally unconscious/unresponsive directly after the ECD discharge without any other explanation (no head trauma, seizure activity, etc). Although it may be possible that the sudden loss of consciousness was not due to the effects of the ECD, it is my opinion that finding a pulse (often radial) could have been spurious during the tumultuous event and that agonal breathing could be mistaken for normal respirations. It is also possible that a pulse and respiration were present initially if the first rhythm was VT before VF, as has been found in the ECD animal studies.[12] In fact, in 1 porcine study,[13] ECD discharge induced a stable monomorphic VT that remained for ≈3 minutes before degeneration to VF. An example of recording an initial pulse and respiration immediately after ECD-induced loss of consciousness was given in the published *New England Journal of Medicine* letter[3] in which a paramedic, present during the entire ECD deployment, felt an initial pulse of 100 (counted for 15 seconds) and respirations of 16,[5] with VF documented by ECG 2 minutes later. Normal breathing has been documented in pigs and sheep for a minute after the onset of ventricular fibrillation and in humans for at least the first 12–15 seconds.[33,34] From these observations, it is clear that continuous monitoring of vital signs and ECG, if available, in an individual unconscious following ECD deployment, should be mandatory.

Conclusions

The animal and clinical data support the conclusion that ECD shocks from a TASER model X26 delivered via probes to the chest can cause cardiac electrical capture. Furthermore, if the capture rate increases sufficiently or if R on T occurs, the development of VF, either directly or via a transition through VT, occurs in animals and, in my opinion, in humans as well. How often this happens is unknown. Although it would seem more likely to occur in individuals exposed to potentially arrhythmogenic drugs, in those who have structural heart disease, and after long or repeated ECD shocks, electrophysiological studies in humans clearly show that only 1 or 2 extra stimuli can provoke VT/VF in particularly susceptible individuals.

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Disclosures

Dr Zipes has served (and in the future may serve) as a paid plaintiff expert witness in ECD-related sudden cardiac arrest/death cases. However, that role has provided access to critical and detailed records necessary to determine the potential for ECD-induced sudden cardiac arrest and death. Despite this conflict, the author has attempted to present the salient facts about the cases and to offer scientific evidence, credible argument, and logic to support the conclusions to a reasonable degree of medical certainty.

References

1. Test results for the Model X26 conducted energy weapon (CEW) in accordance with TASER International device specifications. April 22, 2009. Report A85R9006/9031/9059B1.
2. USA: "Less than lethal"? List of deaths following use of stun weapons in US law enforcement 3 June 2001 to 31 August 2008. Amnesty International Publications. December 16, 2008. Index Number AMR 51/010/2008.
3. Kim PJ, Franklin WH. Ventricular fibrillation after stun-gun discharge: letter to the editor. *N Engl J Med*. 2005;353:958–959.
4. Kroll MW, Calkins H, Luceri RM. Letter to the editor. *J Am Coll Cardiol*. 2007;49:732.
5. Hutchison J. Deposition in Circuit Court of Cook County, 21 June, 2007. *Ortega-Piron v Lopez.* 05 L 1643.
6. Schwarz ES, Barra M, Liao MM. Successful resuscitation of a patient in asystole after a TASER injury using a hypothermia protocol. *Am J Emerg Med.* 2009;27:515.e1–515.e2.
7. Swerdlow CD, Fishbein MC, Chaman L, Lakkireddy DR, Tchou P. Presenting rhythm in sudden deaths temporally proximate to discharge of TASER conducted electrical weapons. *Acad Emerg Med.* 2009;16:726–739.
8. Falk RH, Zoll PM, Zoll RH. Safety and efficacy of noninvasive cardiac pacing: a preliminary report. *N Engl J Med*. 1983;309:1166–1168.
9. Klein LS, Miles WM, Heger JJ, Zipes DP. Transcutaneous pacing: patient tolerance, strength-interval relations, and feasibility for programmed electrical stimulation. *Am J Cardiol*. 1988;62:1126–1129.
10. Grimnes S, Martinsen OG. Clinical applications of bioelectricity. In: *Biomedical Engineering Desk Reference*. 1st ed. New York, NY: Elsevier; 2009:241–382.
11. Lakkireddy D, Wallick D, Ryschon K, Chung MK, Butany J, Martin JD, Saliba W, Kowalewski W, Natale A Tchou PJ. Effects of cocaine intoxication on the threshold for stun gun induction of ventricular fibrillation. *J Am Coll Cardiol*. 2006;48:805–811.
12. Nanthakumar K, Billingsley IM, Masse S, Dorian P, Cameron D, Chauhan VS, Downar ED, Sevaptsidis E. Cardiac electrophysiological consequences of neuromuscular incapacitating device discharges. *J Am Coll Cardiol*. 2006;48:798–804.
13. Dennis AJ, Valentino DJ, Walter RJ, Nagy KK, Winners J, Bokhari F, Wiley DE, Joseph KT, Roberts RR. Acute effects of Taser X26 discharges in a swine model. *J Trauma*. 2007;63:581–590.
14. Walter RJ, Dennis AJ, Valentino DJ, Margeta B, Nagy KK, Bokhari F Wiley DE, Joseph KT, Roberts RR. TASER X26 discharges in swine produce potentially fatal ventricular arrhythmias. *Acad Emerg Med*. 2008; 15:66–73.
15. Valentino DJ, Walter RJ, Dennis AJ, Margeta B, Starr F, MD, Nagy KK, Bokhari F, Wiley DE, Joseph KT, Roberts RR. Taser X26 Discharges in swine: ventricular rhythm capture is dependent on discharge vector. *J Trauma*. 2008;65:1478–1485.
16. Dawes DM, Ho JD, Cole JB, Reardon RF, Lundin EJ, Terwey KS, Falvey DG, Miner JR. Effect of an electronic control device exposure on a methamphetamine intoxicated animal model. *Acad Emerg Med*. 2010;17: 436–443.
17. Cao M, Shinbane JS, Gillberg JM, Saxon LS, Swerdlow CD. Taser-induced rapid ventricular myocardial capture demonstrated by pacemaker intracardiac electrograms. *J Cardiovasc Electrophysiol*. 2008;18:876–879.
18. Ho JD, Dawes DM, Reardon RF, Strote SR, Kunz SN, Nelson RS, Lundin EJ, Orozco BS, Miner JR. Human cardiovascular effects of a new generation conducted electrical weapon. *Forensic Sci Int*. 2011;204:50–57.
19. Wu JY, Sun H, O'Rourke AP, Huebner SM, Rahko PS, Will JA, Webster JG. Taser blunt probe dart-to-heart distance causing ventricular fibrillation in pigs. *IEEE Trans Biomed Eng*. 2008;55:2768–2771.
20. Lakkireddy D, Wallick D, Verma A, Ryschon K, Kowalewski W, Wazni O, Butany J, Martin D, Tchou PJ. Cardiac effects of electrical stun guns: does position of barbs contact make a difference? *Pacing Clin Electrophysiol*. 2008;31:398–408.
21. Dawes DM, Ho JD, Johnson MA, Lundin E, Janchar TA, Miner JR. 15-Second conducted electrical weapon exposure does not cause core temperature elevation in non-environmentally stressed resting adults. *Forensic Sci Int*. 2008;176:253–257.
22. Bozeman WP, Hauda WE 2nd, Heck JJ, Graham DD, Martin BP, Winslow JE. Safety and injury profile of conducted electrical weapons used by law enforcement officers against criminal suspects. *Ann Emerg Med*. 2009;53:480–489.

23. Strote J, Walsh M, Angelidis M, Basta A, Hutson HR. Conductive electrical weapon use by law enforcement: an evaluation of safety and injury. *J Trauma*. 2010;68:1239–1246.
24. Pasquier M, Carron PM, Vallotton L, Yersin B. Electronic control device exposure: a review of morbidity and mortality. *Ann Emerg Med*. 2011; 58:178–188.
25. Nasrallah A, Hall RJ, Garcia E, Kyger ER, Hallman GL, Cooley DA. Runaway pacemaker in seven patients: a persisting problem. *J Thorac Cardiovasc Surg*. 1975;69:365–368.
26. Vilke GM, DeBard ML, Chan TC, Ho JD, Dawes DM, Hall C, Curtis MD, Wysong C, Mash DC, Coffman SR, McMullen MJ, Metzger JC, Roberts JR, Sztajnkrcer MD, Henderson SO, Adler J, Czarnecki F, Heck J, Bozeman WP. Excited delirium syndrome (EXDS): defining based on a review of the literature [published online ahead of print March 24, 2011]. *J Emerg Med*. doi:10.1016/j.jemermed.2011.02.017.
27. Madias C, Fitzgibbons TP, Alsheikh-Ali AA, Bouchard JL, Kalsmith B, Garlitski AC, Tighe DA, Estes NA 3rd, Aurigemma GP, Link MS. Acquired long QT syndrome from stress cardiomyopathy is associated with ventricular arrhythmias and torsades de pointes. *Heart Rhythm*. 2011;8:555–561.
28. Ideker RE, Dosdall DJ. Can the direct cardiac effects of the electrical pulses generated by the TASER X26 cause immediate or delayed cardiac arrest in normal adults? *Am J Forensic Med Path.* 2007;28:195–201.
29. Greenspon AJ, Schaal SF. The "holiday heart": electrophysiologic studies of alcohol effects in alcoholics. *Ann Intern Med*. 1983;98:135–139.
30. *Study of Deaths Following Electro Muscular Disruption: NIJ Report*. Washington, DC: US Department of Justice, Office of Justice Programs; May 2011.
31. Braidwood Hearing and Study Commission report into the death of Robert Dziekanski. www.braidwoodinquiry.ca.
32. Lee BK, Vittinghoff E, Whiteman D, Parka M, Lau LL, Tseng ZH. Relation of Taser (electrical stun gun) deployment to increase in in-custody sudden deaths. *Am J Cardiol*. 2009;103:877–880.
33. Zuercher M, Ewy GA, Hilwig RW, Sanders AB, Otto CW, Berg RA, Kern KB. Continued breathing followed by gasping or apnea in a swine model of ventricular fibrillation cardiac arrest. *BMC Cardiovasc Disord.* 2010;10:36.
34. Haouzi P, Ahmadpour N, Bell HJ, Artman S, Banchs J, Samii S, Gonzalez M, Gleeson K. Breathing patterns during cardiac arrest. *J Appl Physiol.* 2010;109:405–411.

CLINICAL PERSPECTIVE

The safety of electronic control devices (ECDs) has been questioned. This article reports 8 cases of TASER X26 ECD–induced loss of consciousness in which first recorded rhythms were ventricular tachycardia/fibrillation in 6 and asystole (after ≈30 minutes of nonresponsiveness) in 1. An external defibrillator reported a shockable rhythm in 1 case, but no recording was made. From an analysis of the cases and data from animal and clinical ECD studies, it is the opinion of the author that ECD stimulation can cause cardiac electrical capture and provoke cardiac arrest resulting from ventricular tachycardia/ventricular fibrillation. The cause is probably ventricular capture during the vulnerable period of the previous beat or ventricular capture at rates too fast for ventricular activation to remain organized. After prolonged ventricular tachycardia/ventricular fibrillation without resuscitation, asystole develops. The purpose of this article is not to condemn ECD use by trained professionals but to make ECD users aware that cardiac arrest caused by ventricular fibrillation can result from an ECD shock and to encourage users to be judicious concerning how and when to use the ECD weapon, to avoid chest shocks if possible, to monitor the person after an ECD shock, and to suspect this adverse response in any victim who loses consciousness. Users should be prepared to resuscitate, including deployment of an automated external defibrillator if indicated.

Sudden Cardiac Arrest and Death Following Application of Shocks From a TASER Electronic Control Device

Douglas P. Zipes

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Correction

In the article by Zipes, "Sudden Cardiac Arrest and Death Following Application of Shocks From a TASER Electronic Control Device," which was published in the May 22, 2012 issue of the journal (*Circulation*. 2012;125:2417–2422), two points are in error.

First, where it is stated that "Individuals were previously clinically healthy males . . ." it was meant that they were clinically healthy from a cardiovascular standpoint without manifest cardiovascular symptoms. As indicated in the table of the original manuscript, autopsy may have shown underlying cardiac disease not clinically manifest. Four were inebriated, as indicated by the drug screen (table), with a history of alcohol abuse; case 3 had an attention deficit disorder, possibly bipolar; case 5 was mentally confused, perhaps due to a post ictal state after a seizure; and case 6 was agitated with a diagnosis of depressive disorder, schizophrenia and medication noncompliance. Second, the first available ECG shown in the figure was a representative ECG strip chosen from those recorded at the time of attempted resuscitation and was not the very first ECG recorded in all cases. Upon comparison of these tracings, there is no material difference in the recorded rhythms. None of these statements change the author's opinion expressed in the conclusion of the article but are presented for completeness and accuracy.

(*Circulation*. 2012;126:e27.)

***Circulation* is available at http://circ.ahajournals.org** **DOI: 10.1161/CIR.0b013e31826488fa**

Correction

In the article by Zipes, "Sudden Cardiac Arrest and Death Following Application of Shocks From a TASER Electronic Control Device," which appeared in the May 22, 2012 issue of the journal (*Circulation*. 2012;125:2417-2422), Dr Zipes did not acknowledge the contributions of Atty. John Burton, Dr Kamaraswamy Nanthakumar, Dr John Miller, and Ms Joan Zipes. The current online version of the letter has been corrected.

(***Circulation*. 2013;127:e839.**)

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In this study, we sought to examine the effects of a CEW application in resting adult volunteers to determine if there was evidence of induced electrical dysrhythmia or direct cellular damage that would indicate a causal relationship between the application of the device and in-custody death (ICD).

METHODS

Study Design

This was a prospective observational study of resting adult volunteers recruited at a TASER International training course in April 2005. The institutional review board of Hennepin County Medical Center approved the study. Subjects provided informed consent before enrollment.

Study Setting and Population

This study was performed with volunteer subjects. As part of their training course, they were to receive a five-second CEW application as required by the course curriculum. All adult subjects (age older than 18 years) were eligible for enrollment. They did not have to participate in the study as a requirement for successful course completion, but declining to participate in the study did not absolve them from receiving the CEW application. The application consisted of a five-second application with projectile darts powered by the TASER X26 model CEW (TASER International) (Figure 1).

Study Protocol

Before undergoing the CEW application, 32 of the 66 subjects were randomly selected to also undergo a baseline electrocardiographic evaluation. Random selection of these subjects was made by taking the next subject in line when an electrocardiography machine became available. All subjects had blood drawn before the CEW application for baseline laboratory analysis for electrolytes, serum markers of skeletal and cardiac muscle injury, and renal function. All subjects completed a medical questionnaire for the purpose of gathering additional medical information for descriptive reporting. The descriptive data points gathered for all subjects included age, gender, medical history, current medications, and significant exertion within the 24 hours before the study.

The CEW application consisted of a standard TASER X26 deployment of probes made from a distance of approximately 7 ft. The subject faced away from the deploying instructor and was supported by assisting personnel per the manufacturer's training recommendations. When the CEW was deployed and muscular incapacitation was achieved, the support personnel would allow the subject to begin to fall to the ground and would assist them to the ground in whichever direction they were falling. The CEW was allowed to run for a standard five-second cycle. On completion of the application, the probes were removed, the probe entry points were disinfected, and adhesive bandages were applied if needed.

Immediately following the CEW application, all subjects underwent repeat venipuncture. This was performed again at 16 and 24 hours after the application. Similarly, the electrocardiography subgroup underwent additional electrocardiography at the same time intervals.

Collected blood samples were analyzed for troponin, myoglobin, lactate, potassium, glucose, blood urea nitrogen, creatinine, and creatine kinase levels. Venipuncture was performed by an independent laboratory organization (Laboratory Corporation of America, Phoenix, AZ) using certified phlebotomists and routine venipuncture practices. After each venipuncture, the specimens were labeled and transported according to the laboratory standard for analysis at an off-site facility. Analysis was performed using standard assays and laboratory instruments (Abbott Diagnostics, Abbott Park, IL).

The subjects selected to participate in the 12-lead electrocardiographic evaluation were connected to cardiac monitors (Medtronic, Minneapolis, MN). The leads were placed in standard configuration on the chest wall, arms,

Figure 1. Cutaway image of TASER X26 conducted electrical weapon. Photograph courtesy of TASER International.

and legs. The computer assessment function was turned off to prevent printing of the assessment on the electrocardiogram to ensure unbiased evaluation. The electrocardiograms were sent to a blinded, independent cardiologist in random order and individually evaluated for abnormalities.

Data Analysis

Data were entered in an Excel (Microsoft Corp., Redmond, WA) database for analysis. Data analysis was performed using Stata 6.0 (Stata Corp., College Station, TX). Descriptive statistics were used when appropriate. The percentage change from baseline laboratory values is presented with 95% confidence intervals (95% CI). Changes were considered insignificant when the confidence interval crossed zero.

RESULTS

A total of 66 subjects were enrolled (65 men and one woman), with a mean (±SD) age of 40.3 (±6.8) years (range, 29–55 years). No eligible subjects were excluded from participation. The mean (±SD) body mass index was 28.4 (±3.5) kg/m^2 (range, 21.1–36.8 kg/m^2). Of the 66 subjects, 51 (77.3%) reported no significant medical history, six (9.1%) reported a history of known hypertension, and six (9.1%) reported hypercholesterolemia. Additionally, one subject reported previous myocardial infarction with triple coronary artery bypass grafting and significant coronary artery disease. One subject also reported a history of congestive heart failure (currently well controlled), another reported a history of a transient ischemic attack two years prior, and one subject reported type 2 diabetes mellitus (Table 1). There were eight reports (12.1%) of a significant family history of coronary artery disease (close relative with early onset of disease). There were also 17 reports (25.8%) of strenuous physical exertion on the day of study enrollment. The strenuous physical exertion described was typical of an exertional aerobic workout or anaerobic weight lifting.

Laboratory results are shown in Table 2. There was no significant change from baseline for serum electrolyte levels and the blood urea nitrogen/creatinine ratio. An increase in serum bicarbonate and creatine kinase levels was noted at 16 and 24 hours. An increase in serum lactate level was noted immediately after exposure that decreased at 16 and 24 hours. Serum myoglobin level was increased from baseline at all three time points. The troponin I levels were all <0.3 ng/mL, except a single value of 0.6 ng/mL in a single subject at the 24-hour post-exposure period. This subject was evaluated in a hospital by a cardiologist, and no clinical evidence of acute myocardial infarction was identified and no evidence of cardiac disability was demonstrated. The troponin I value returned to normal within eight hours of its reported elevation. The subject was never symptomatic and continued a regimen of daily aerobic exercise after the hospital evaluation without difficulty.

Thirty of the 32 electrocardiograms were interpreted as normal. The two abnormal electrocardiograms remained the same at all four time points (one was left ventricular hypertrophy, and one was an occasional sinus pause). No other abnormalities were noted.

Table 1
Summary of Subject Medical Histories

Medical History		*n*	Percentage of Total
Past history	None	51	77.3
	Hypertension	6	9.1
	Hypercholesterolemia	6	9.1
	Mitral valve prolapse	2	3.0
	Hypothyroidism	1	1.5
	Congestive heart failure	1	1.5
	Previous myocardial infarction	1	1.5
	Cerebrovascular disease	1	1.5
	Asthma	1	1.5
	Diabetes	1	1.5
	Gout	1	1.5
Medications	None	52	78.8
	Statin	6	9.1
	Antihypertensive	6	9.1
	Aspirin	6	9.1
	Thyroxine	2	3.0
	Glipizide	1	1.5
	Allopurinol	1	1.5
	Nitroglycerin	1	1.5
Family history	None	44	66.7
	Coronary artery disease	8	12.1
	Diabetes	9	13.6
	Other*	13	19.7

* All other reported conditions (hypertension, hypercholesterolemia, stroke, abdominal aortic aneurysm, implanted cardiac pacemaker, rheumatoid arthritis, various cancers).

DISCUSSION

Conducted electrical weapons are considered to be an intermediate weapon by law enforcement agencies (intermediate weapons are those devices that generally can induce subject compliance due to pain or incapacitation and are a level above empty-hand control techniques but less than deadly force). Examples of intermediate weapons include devices such as aerosolized chemical irritants, impact batons, and projectile beanbags. TASER is a brand name (acronym for Thomas A. Swift Electric Rifle) of CEW. The terms "TASER device" and "CEW" are often used interchangeably because, at the time of this writing, there are no other CEW manufacturers that have brought products to market. Currently, TASER International manufactures two law enforcement models (X26 and M26) and three civilian models (X26^c, M18, and M18L). The X26 is the latest generation and the most popular model currently in use and was the model used in this study. It is considered to be a nonlethal weapon under the definition set forth by the U.S. Department of Defense.[7]

The X26 is programmed to deliver a roughly rectangular pulse of approximately 100-microsecond duration with about 100 μC of charge at 19 pulses per second for five seconds.[8] The peak voltage across the body is approximately 1,200 V, but the weapon also develops an open-circuit arc of 50,000 V to traverse clothing in cases where no direct contact is made. The average current is approximately 2.1 mA. It uses compressed nitrogen to fire two metallic darts up to a maximum of 35 ft with a

Table 2
Summary of Serum Analysis for Study Subjects

	Baseline	Time 2	Time 3	Time 4
Glucose (mg/dL)				
Mean	94.5	98.5	92.1	101.6
SD	13.8	16.5	22.6	19.4
Range	56–137	44–161	51–185	69–151
% Change (95% CI)		4.9 (−1.1, 8.6)	−1.0 (−7.6, 5.6)	7.6 (−0.5, 14.8)
BUN (mg/dL)				
Mean	16.5	16.5	16.9	17.2
SD	4.5	4.4	4.1	3.5
Range	9–31	9–29	11–31	11–28
% Change (95% CI)		−0.7 (−2.8, 1.4)	−5.3 (−10.2, 0.4)	−5.4 (−11.0, 0.2)
Creatinine (mg/dL)				
Mean	1.1	1.1	1.1	1.1
SD	0.16	0.15	0.13	0.14
Range	0.7–1.5	0.7–1.4	0.7–1.4	0.7–1.5
% Change (95% CI)		−1.0 (−2.5, 0.5)	2.1 (−0.7, 4.8)	−1.4 (−5.3, 2.6)
BUN/creatinine ratio				
Mean	15.1:1	15.0:1	16.0:1	15.3:1
SD	3.5	3.5	3.7	2.9
Range	9–24:1	9–27:1	9–26:1	11–25:1
% Change (95% CI)		0.0 (−2.2, 2.2)	8.1 (3.4, 12.9)	4.7 (−0.2, 9.5)
Sodium (mmol/L)				
Mean	138.8	138.9	137.4	137.8
SD	2.2	2.2	1.9	2.4
Range	135–148	134–147	134–142	134–145
% Change (95% CI)		−0.1 (−0.4, 0.2)	1.0 (0.5, 1.4)	0.7 (0.2, 1.1)
Potassium (mmol/L)				
Mean	4.1	3.9	4.5	4.2
SD	0.3	0.4	0.4	0.3
Range	3.5–5.0	3.3–4.9	3.7–5.7	3.2–5.2
% Change (95% CI)		4.1 (1.9, 6.4)	−8.7 (−11.7, −5.6)	−2.2 (−4.6, 0.1)
Chloride (mmol/L)				
Mean	100.3	99.9	101.1	101.0
SD	2.1	2.0	2.5	2.7
Range	96–106	95–109	96–108	96–108
% Change (95% CI)		0.4 (0.0, 0.8)	−0.7 (−1.4, 0.0)	−0.6 (−1.3, 0.0)
Bicarbonate (mmol/L)				
Mean	22.6	22.0	24.6	23.8
SD	1.9	2.1	2.1	2.3
Range	19–26	18–27	19–29	18–29
% Change (95% CI)		2.4 (0.4, 4.3)	−9.1 (−11.8, −6.3)	−5.0 (−7.2, −2.8)
Calcium (mg/dL)				
Mean	9.9	9.9	9.9	9.9
SD	0.3	0.3	0.3	0.3
Range	9.3–10.7	9.0–10.6	9.2–10.7	9.1–10.6
% Change (95% CI)		0.3 (−0.4, 0.9)	−0.4 (−1.0, 0.3)	0.1 (−0.8, 0.9)
Creatine kinase (U/L)				
Mean	185.1	184.1	221.6	242.3
SD	99.4	99.8	143.9	170.5
% Change (95% CI)		0.9 (−0.5, 2.2)	−23.9 (−38.1, −9.8)	−32.2 (−49.3, −15.0)
Range	71–479	60–484	50–806	52–909
Troponin I (ng/mL)				
Mean	0	0	0	0
SD	0	0	0	0
Range	0	0	0	0
% Change (95% CI)		0	0	0
Lactate (mg/dL)				
Mean	15.8	24.7	18.3	19.8
SD	5.7	7.6	6.8	6.7
Range	7–44	9–45	7–36	9–37
% Change (95% CI)		−66.9 (−80.8, −53.0)	−22.3 (−35.1, −9.5)	−30.8 (−43.6, −17.9)
Myoglobin (ng/mL)				
Mean	32.4	45.5	42.9	51.3
SD	15.1	27.1	22.4	29.8
Range	11–100	15–167	18–130	17–61
% Change (95% CI)		−34.1 (−57.4, −10.7)	−36.3 (−47.3, −25.6)	−64.0 (−89.4, −38.6)

Percent change is from baseline.

predetermined angled rate of spread. It is capable of transmitting an electrical impulse through two cumulative inches of clothing. When it makes adequate contact and the darts are of adequate separation, it causes involuntary contractions of the regional skeletal muscles that render the subject incapable of voluntary movement. If the darts are fired at very close range and do not achieve adequate separation, full muscular incapacitation may not be achieved, and the device is then used to encourage certain behavior through pain compliance. Additionally, the TASER device has electrical contact points at its tip that are approximately 1.5 inches apart. These contact points may be touched to a subject during discharge of the weapon and are also considered a pain compliance technique because the separation is not adequate to cause a full, involuntary contraction of muscles.

It has been theorized that CEWs have been associated with several sudden and unexpected subject deaths while in law enforcement custody. This ICD phenomenon is not new, and similar phenomena have been described in psychiatric literature dating back to the mid-1800s.[9] Over the years, there have been attempts to link ICD with single causative factors, such as use of chemical irritants (e.g., pepper spray), restraint and positional asphyxia, structural cardiac abnormalities, or use of illicit stimulant medication.[10–22] Many of these links have been questioned, disproved, or found to be absent.[23–29] This has generated more questions than answers in the search for a common cause.

One theory that has gained some recent popularity has been that of the condition of excited delirium and metabolic acidosis.[30] The described features of excited delirium syndrome include agitation, incoherence, hyperthermia, paranoia, inappropriate and often violent behavior, constant motion, and feats of incredible strength. This syndrome is closely associated with sudden, unexpected death.[19,29] It is believed that precursors to this condition are chronic, illicit stimulant abuse, presence of certain mental health conditions, and also use of certain mental health medications. It is believed that the state of excited delirium sets the stage for the subject to enter a metabolic acidosis condition, and this can be profound.[31] If left untreated, the subject will become acidotic to a point that is not compatible with life and will experience a cardiorespiratory arrest. Surveillance data seem to correlate this type of behavior with those at highest risk for an ICD event.[3]

However, another more recent theory is that of the TASER-induced ICD (TIICD). It is a perception by many that because a CEW incapacitates through the generation of electricity, it is somehow causing death, presumably from an electrically induced fatal arrhythmia. There have been media sources that have incorrectly compared CEWs with the electric chair used in capital punishment, although the electrical current specifications for each are markedly different.[32] If the TIICD theory is correct, it would be expected that electrically induced fatal arrhythmias would be inducible in the laboratory setting. This has not been the case, and there is evidence to show that the current, available CEW output would need to be increased to a minimum of 15 times its current setting to reliably induce ventricular fibrillation in a 60-lb animal.[33] This same study showed that animals with heavier masses required even greater outputs. Additionally, there have been instances when persons of small stature have experienced a CEW deployment without evidence of sudden death.[34,35] Collectively, these data do not support the theory of a CEW-induced fatal arrhythmia as the cause of ICD events.

If TIICD were to occur in real life, it would be expected that any induction of arrhythmia would be instantaneous and result in instantaneous collapse and cardiac arrest. However, in a surveillance of eight months of ICD events in the United States, only 27% of ICD events were associated with occurrence proximal to application of a CEW, and in none of these cases did the person collapse instantaneously after the application.[3] There have been two other data sources that seem to counter the TIICD theory. The first was a small study by Levine et al. that measured cardiac rhythm strips before, during, and after CEW application on a pool of volunteers. The conclusion was that the CEW application did nothing to create an abnormality on the observed rhythm strip.[36] The second and more compelling set of data comes from the training classes conducted by CEW manufacturer TASER International. These classes have delivered more than 100,000 CEW applications to participants with no reported collapses, cardiac arrests, or fatalities.[8] Additionally, the results of our study do not support this theory.

There has been one report in a letter to the editor of a medical journal of a case of ventricular fibrillation after exposure to a CEW.[37] Upon review of the paramedic field report, the subject received a CEW application because of apparent threatening behavior toward a police officer during a prolonged, agitated state. The subject was successfully subdued but found to be in cardiorespiratory arrest approximately 14–23 minutes after the CEW application. We believe that this case is very similar to every other described in the literature in which the ICD event occurs proximal to CEW exposure but collapse is not instantaneous. We believe that the facts of this case report do not support an electrically induced dysrhythmia.

Because the TIICD theory does not seem to be consistent with an instantaneously induced catastrophe, it has also been theorized that perhaps the application of the CEW somehow causes a more insidious, longer-term problem that manifests itself minutes or hours after the event. It is hypothesized that this could take the form of a silent myocardial event. It is also thought that perhaps the CEW application could induce rhabdomyolysis that has been associated with an excited delirium condition.[38] Because of this theory, we undertook this project.

Using standard laboratory analyses and electrical cardiac monitoring devices, we were unable to demonstrate a significant change from baseline in standard serum electrolyte values of the test subjects after application of the CEW. Of note, it is theorized that an association between CEW application and ICD is due to a possible induced hyperkalemia from cellular damage and necrosis. Our findings do not support this, and the mean serum potassium value actually decreased slightly immediately after CEW exposure. Additionally, we did not demonstrate any decrease in serum bicarbonate levels that would lead to a suggestion of induced acidosis.

With regard to serum markers of muscle injury such as creatine kinase, lactate dehydrogenase, and myoglobin,

we did demonstrate increased levels after CEW exposure. These findings were not unexpected based on previous literature demonstrating elevated levels of skeletal muscle damage markers for at least 48 hours following an exertional event.[39] We consider these CEW data to be a baseline with regard to resting human subjects. A possible consideration is that in subjects who experience an ICD event, there may be a connection between their hypermetabolic and presumed acidosis state (due to fleeing, fighting, stimulant use, and so on) and the application of CEWs. This possible connection has not yet been shown or disproved and remains as an area requiring further investigation.

The single subject with the slight elevation of troponin I level was initially concerning. All of this subject's troponin levels remained within the normal range (laboratory reference normal is 0.0–0.4 ng/mL) except for the single level drawn at 24 hours after CEW exposure. This level was 0.6 ng/mL. It should be noted that this subject was a very fit and athletic individual and had performed a rigorous aerobic workout regimen without difficulty about three hours before the CEW exposure. Despite being asymptomatic and feeling well, he was immediately taken to a hospital where he underwent admission and extensive cardiac evaluation from a group of consulting cardiologists. His initial troponin level at the hospital was drawn within eight hours of his elevated level and was 0.1 ng/mL. His inpatient evaluation included a treadmill stress test (Treadmill Myoview test utilizing standard Bruce protocol with a double product of 24,335 achieved) and a rest/adenosine-augmented stress gated tomographic myocardial perfusion study utilizing Tc99m radiopharmaceutical injection. The results of both tests were interpreted as normal. There were several explanations offered as possible causes by the consulting cardiologists. These included laboratory error, delayed clearance of troponin related to subject baseline physiology, or idiopathic and indeterminate etiology. There was agreement that there was no indication of myocardial damage or ischemia, and the subject was allowed to return to regular duty without limitations.

LIMITATIONS

Previous studies conducted on CEWs have used police volunteers as study subjects. The criticisms surrounding this type of sampling have focused on the perceived health of the study population. It has been stated that persons in the police profession have above-average health and fitness when compared with other members in society. Therefore, studies based on this population might be biased. The same criticism could be brought forward in this study. However, we collected the health histories of all of the volunteers and found that a surprising number of the volunteers had significant health problems as previously outlined. Based on this, we believe that this study population encompasses volunteers with health issues not unlike the general population. Additionally, the mean body mass index calculated for the study group places them in the "overweight" category for American adults. We acknowledge that the study population did not have a diagnosed mental illness condition and did not have an apparent history of illicit stimulant abuse. Both of these conditions are recognized to be present in a high percentage of subjects who experience an ICD event.[3,40] However, we also recognize that a real-time study of this population is unlikely to meet any protections required for human subject studies in this country.

Another limitation to this study is that the population sampled was considered to be at rest. This is unlike the population that meets the profile at high risk for an ICD event.[3] We recognize this but believe that these baseline data make an important contribution to this area of study, and we recommend that further study be conducted with other types of sample populations to determine associative risk, if any.

An additional criticism of this project might be that the study subjects received only a five-second application from the CEW. We recognize that there are occasional reports of multiple applications or prolonged exposures to CEWs, but we designed the study around the most common time exposure reported. The period of five seconds was used because the manufacturer's collected data suggest that a majority of field applications (67%) are for five seconds or less.[8]

CONCLUSIONS

In this resting adult population, the TASER X26 CEW did not affect the recordable cardiac electrical activity within a 24-hour period following a standard five-second application. We were unable to detect any induced electrical dysrhythmias or significant direct cardiac cellular damage that may be related to sudden and unexpected death proximal to CEW exposure. Additionally, we did not demonstrate evidence of dangerous hyperkalemia or induced acidosis. We recommend further study in the area of the ICD phenomenon to better understand its causes.

References

1. American Civil Liberties Union. Citing deaths in police custody, ACLU of Colorado calls for limits on use of electroshock weapons. ACLU Library, Feb 26, 2004. Available at: http://www.aclu.org/CriminalJustice/CriminalJustice.cfm?ID=15167&c=15. Accessed Nov 1, 2005.
2. Amnesty International. Excessive and lethal force? Amnesty International's concerns about deaths and ill-treatment involving police use of TASERs. Amnesty International Library, Nov 30, 2004. Available at: http://web.amnesty.org/library/index/engamr511392004. Accessed Nov 1, 2005.
3. Ho JD, Reardon RF, Heegaard WG. Deaths in police custody: an 8 month surveillance study [abstract]. Ann Emerg Med. 2005; 46(Suppl):S94.
4. Berenson A, Demands rise for tighter oversight on use of stun guns. New York Times. Feb 17, 2005:A24.
5. Anglen R. Taser safety claim questioned. Arizona Republic, Jul 18, 2004. Available at: http://www.azcentral.com/specials/special43/articles/0718taser-main18.html). Accessed Nov 1, 2005.
6. Anglen R. 73 cases of death following stun gun use. Arizona Republic, Oct 12, 2004. Available at: http://

www.azcentral.com/specials/special43/articles/0915taserlist16-ON.html. Accessed Nov 1, 2005.
7. Anonymous. Department of Defense Directive 3000.3 Policy for Nonlethal Weapons. United States Department of Defense. Jul 9, 1996. Available at: http://www.dtic.mil/whs/directives/corres/pdf/d30003_070996/d30003p.pdf. Accessed Nov 1, 2005.
8. TASER training video and information disk, version 12. Scottsdale, AZ: TASER International, Nov 2004.
9. Bell L. On a form of disease resembling some advanced stages of mania and fever, but so contradistinguished from any ordinary observed or described combination of symptoms as to render it probable that it may be overlooked and hitherto unrecorded malady. Am J Insanity. 1849; 6:97–127.
10. American Civil Liberties Union of Southern California. Pepper spray update: more fatalities, more questions. Pepper Spray Update, Jun 1995. Available at: http://www.aclu-sc.org/attachments/p/Pepper_Spray_New_Questions.pdf. Accessed Nov 1, 2005.
11. Stratton SJ, Rogers C, Brickett K, Gruzinski G. Factors associated with sudden death of individuals requiring restraint for excited delirium. Am J Emerg Med. 2001; 21:187–91.
12. Eckart RE, Scoville SL, Campbell CL, et al. Sudden death in young adults: a 25-year review of autopsies in military recruits. Ann Intern Med. 2004; 141: 829–35.
13. Maron BJ, Roberts WC, Epstein SE. Sudden death in hypertrophic cardiomyopathy: a profile of 78 patients. Circulation. 1982; 65:1388–94.
14. Ross DL. Factors associated with excited delirium deaths in police custody. Mod Pathol. 1998; 11: 1127–37.
15. Reay DT, Fligner CL, Stilwell AD, Arnold J. Positional asphyxia during law enforcement transport. Am J Forensic Med Pathol. 1992; 13:90–7.
16. O'Halloran RL, Lewman LV. Restraint asphyxia in excited delirium. Am J Forensic Med Pathol. 1993; 13: 289–95.
17. O'Halloran RL, Frank JG. Restraint asphyxia. Am J Forensic Med Pathol. 2000; 21:420–2.
18. O'Halloran RL, Frank JG. Asphyxial death during prone restraint revisited: a report of 21 cases. Am J Forensic Med Pathol. 2000; 21:39–52.
19. Wetli CV, Mash D, Karch SB. Cocaine-associated agitated delirium and the neuroleptic malignant syndrome. Am J Emerg Med. 1996; 14:425–8.
20. Karch SB, Stephens BG. Drug abusers who die during arrest or in custody. J R Soc Med. 1999; 92: 110–3.
21. Pestaner JP, Southall PE. Sudden death during arrest and phencyclidine intoxication. Am J Forensic Med Pathol. 2003; 24:119–22.
22. Ruttenber AJ, Lawler-Heavner J, Yin M, Wetli CV, Hearn WL, Mash DC. Fatal excited delirium following cocaine use: epidemiologic findings provide new evidence for mechanisms of cocaine toxicity. J Forensic Sci. 1997; 42:25–31.
23. United States Department of Justice, Office of Justice Programs, National Institute of Justice. The effectiveness and safety of pepper spray. Research for Practice, Apr 2003. Available at: http://www.ncjrs.gov/pdffiles1/nij/195739.pdf. Accessed Nov 1, 2005.
24. Chan TC, Vilke GM, Clausen J, et al. The effect of oleoresin capsicum "pepper" spray inhalation on respiratory function. J Forensic Sci. 2002; 47:299–304.
25. Chan TC, Vilke GM, Neuman T, Clausen JL. Restraint position and positional asphyxia. Ann Emerg Med. 1998; 32:116–8.
26. Chan TC, Vilke GM, Neuman T. Reexamination of custody restraint position and positional asphyxia. Am J Forensic Med Pathol. 1998; 19:201–5.
27. Chan TC, Neuman T, Clausen J, Eisele J, Vilke GM. Weight force during prone restraint and respiratory function. Am J Forensic Med Pathol. 2004; 25:185–9.
28. Kornblum RN, Reddy SK. Effects of the TASER in fatalities involving police confrontation. J Forensic Sci. 1992; 37:956–8.
29. Glatter K, Karch SB. Positional asphyxia: inadequate oxygen, or inadequate theory? Forensic Sci Int. 2004; 141:201–2.
30. Di Maio TG, Di Maio VJM. Excited Delirium Syndrome Cause of Death and Prevention, ed 1. Boca Raton, FL: Taylor & Francis, 2006.
31. Hick JL, Smith SW, Lynch MT. Metabolic acidosis in restraint-associated cardiac arrest: a case series. Acad Emerg Med. 1999; 6:239–43.
32. Anonymous. Taser packs potent but brief punch of electricity. USA Today. Jun 3, 2005:A13.
33. McDaniel WC, Stratbucker RA, Nerheim M, Brewer JE. Cardiac safety of neuromuscular incapacitating defensive devices. PACE. 2005; 28(Suppl):S284–7.
34. United Press International. Police use stun gun on 6 year old. The Washington Times. Nov 12, 2004. Available at: http://washingtontimes.com/upi-breaking/20041112-111756-3329r.htm. Accessed Nov 1, 2005.
35. Police review policy after tasers used on kids. CNN, Nov 15, 2004. Available at: http://www.cnn.com/2004/US/11/14/children.tasers/). Accessed Nov 1, 2005.
36. Levine SD, Sloane C, Chan T, Vilke G, Dunford J. Cardiac monitoring of subjects exposed to the TASER [abstract]. Acad Emerg Med. 2005; 12(Suppl 1):71.
37. Kim PJ, Franklin WH. Ventricular fibrillation after stun-gun discharge [letter]. N Engl J Med. 2005; 353: 958–9.
38. Ruttenber AJ, Mcanally HB, Wetli CV. Cocaine-associated rhabdomyolysis and excited delirium: different stages of the same syndrome. Am J Forensic Med Pathol. 1999; 20:120–7.
39. French DN, Kraemer WJ, VanHeest JL, et al. Physiological damage and stress of a competitive NCAA division 1 football game. Med Sci Sports Exerc. 2003; 35(Suppl):S320.
40. Pollanen MS, Chiasson DA, Cairns JT, Young JG. Unexpected death related to restraint for excited delirium: a retrospective study of deaths in police custody and in the community. CMAJ. 1998; 158:1603–7.

Can TASER Electronic Control Devices Cause Cardiac Arrest?

TASER Electronic Control Devices Can Cause Cardiac Arrest in Humans

Douglas P. Zipes, MD

The TASER X26 electronic control device (ECD) is a handgun-shaped device that uses compressed nitrogen to fire darts ranging from 9 to 14 mm in length that impale the clothes or skin of an individual up to a distance of 35 ft. Wires connect the darts to the device. The TASER X26 functions as a constant current generator and delivers an initial 50 000-V to begin an arcing shock (the actual voltage delivered to the body is in the range of 1400–2520 V), followed by electric pulses of 105- to 155-microsecond duration, at a frequency of ≈19 Hz (≈1140 times per minute), and 80- to 125-microcoulomb delivered charge.[1] A single trigger pull discharges a 5-second cycle that can be shortened by a safety switch to deactivate the device or prolonged if the trigger pull is held. The trigger can be activated multiple times. The X26 data port stores the time and date of use and number and duration of trigger pulls. If effective, the shock elicits neuromuscular inhibition, allowing law enforcement to gain control of a suspect (see www.youtube.com/watch?v=ACUjnJBHIZc for a TASER demonstration). The device can also be applied in a "drive-stun" mode by directly pressing the X26 ECD against the skin to achieve pain compliance without neuromuscular inhibition. The TASER X26 is the most widely sold ECD. Called a less lethal or nonlethal weapon because it is supposed to be deployed to temporarily incapacitate, not to kill the subject, the X26 is not considered a firearm and therefore is not regulated by the Bureau of Alcohol, Tobacco, Firearms and Explosives.

Response by Kroll et al on p 111

Purpose

The purpose of this article is to present information to support the conclusion that the TASER X26 ECD can cause cardiac arrest in humans. As noted in an earlier article,[2] the purpose is not to offer an opinion about whether the use of TASER or any other ECD product is appropriate because I think that decision belongs to trained law-enforcement professionals, not physicians.

Background

A previous publication[2] presented 8 cases of sudden cardiac arrest that, in my opinion, resulted from delivery of electric impulses generated by a TASER X26 ECD. None had manifest cardiovascular symptoms, although several had non–cardiac-related medical problems, including alcohol abuse, attention deficit disorder, mental confusion that was possibly postictal from a seizure, and depression/schizophrenia. At autopsy, several were alleged to have had underlying heart disease (Table). All had rapid loss of consciousness after X26 deployment and ECD shocks via 1 or more darts in the anterior chest (Figures 1 and 2). Selected ECGs recorded at various time intervals during resuscitation attempts showed ventricular tachycardia (VT)/ventricular fibrillation (VF) in 5, a shockable rhythm by an automated external defibrillator in

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This article is Part II of a 2-part article. Part I appears on p 93.

The online-only Data Supplement is available with this article at http://circ.ahajournals.org/lookup/suppl/doi:10.1161/CIRCULATIONAHA.113.005504/-/DC1.

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Table. Summary of the 8 Cases Reported as Having Cardiac Arrest After X26 Administration

Case	Age, y	Height/ Weight, lb	Length of ECD Shock(s), s	Response to ECD Shock	Time to Initial ECG After ECD Shock, min	Initial Recorded Rhythm	Drug Screen	Cardiac Findings at Autopsy	Comments
1	48	6 ft 0 in/155	5, 8, 5	LOC toward end of last ECD cycle	Several	VT/VF	BAC 0.35 g/100 mL; THC present	Survived with memory impairment; normal echocardiogram	5 AED shocks, intravenous epinephrine, and lidocaine eventually restored a perfusing rhythm
2	17	5 ft 7 in/170	37, 5	LOC toward end of a 37-s cycle	>4.5	VF	Negative	410 g; focal atherosclerosis; plaintiff pathologist: normal; defense pathologist: HCM	3 defibrillating shocks and an additional 3 shocks from a second AED at least 9 min after the collapse failed to resuscitate
3	17	5 ft 8 in/115	5	ILOC	>5	VF	BAC 0.25 g/100 mL; THC present	270 g; normal heart	Asystole developed after the AED shock and then PEA; subsequently, VF recurred and a second AED shock was delivered, followed by asystole/ PEA; could not be resuscitated
4	24	5 ft 10 in/176	11	ILOC	≈10	AED: "shockable rhythm"; asystole after shock; no recordings available	BAC 0.319 g/100 mL	400 g; plaintiff pathologist: no specific pathology; defense pathologist: lymphocytic myocarditis	Said to be breathing initially with a weak radial pulse; resuscitated in hospital; life support withdrawn after 3 d because of anoxic encephalopathy
5	33	6 ft 2 in/220	13 shocks totaling 62 s in <3 min	LOC toward the end of multiple shocks	≈13	Fine VF vs asystole	Gabapentin 31 μg/mL	470 g; 10%–20% narrowing of the LAD; normal histology	Gabapentin taken for seizure disorder
6	24	5 ft 6 in/144	49, 5	LOC toward end of 49-s shock	≈10	VT/VF	Negative	366.7 g; normal gross and microscopic findings	Said to be breathing initially; could not be resuscitated
7	16	5 ft 3 in/130	5	ILOC	≈10	VT/VF	THC	380 g; medical examiner diagnosis: right ventricular cardiomyopathy, disputed by plaintiff's expert	6 AED shocks for VT/VF resulted in asystole/PEA; could not be resuscitated
8	23	5 ft 9 in/173	21, 7, 3	LOC toward end of 21-s shock	≈30	Asystole	BAC 0.111 g/100 mL	400 g; mild interstitial fibrosis of compact atrioventricular node; interstitial fibrosis, atrophy, and vacuolization of penetrating and branching bundle	Said to be breathing with pulse initially; could not be resuscitated; cardiac pathologist could not determine whether changes contributed to death

AED indicates automated external defibrillator; BAC, blood alcohol concentration; ECD, electronic control device; HCM, hypertrophic cardiomyopathy; ILOC, immediate loss of consciousness during/after initial shock; LAD, left anterior descending coronary artery; LOC, loss of consciousness during/after initial shock; PEA, pulseless electric activity; THC, tetrahydrocannabinol, positive screen for marijuana; VF, ventricular fibrillation; and VT, ventricular tachycardia. Heart weight is given in grams. Gabapentin is Neurontin.

Reproduced from Zipes DP. Sudden cardiac arrest and death following application of shocks from a TASER electronic control device. *Circulation.* 2012;125:2417–2422.[2]

1 (no ECG recording), fine VF/asystole in 1, and asystole in 1 (Figure 3). The last 2 cases had significant time delays from X26 deployment and loss of consciousness until ECGs were recorded (Figures 4 and 5). Only 1 of 8 was resuscitated but with residual anoxic cognitive impairment.

In an accompanying editorial, Myerburg et al[3] stated that the article established "proof of concept" and that the information in at least 2 of the cases lent "…credence to the likelihood of an association that is strong enough to demonstrate a cause-and-effect relationship."

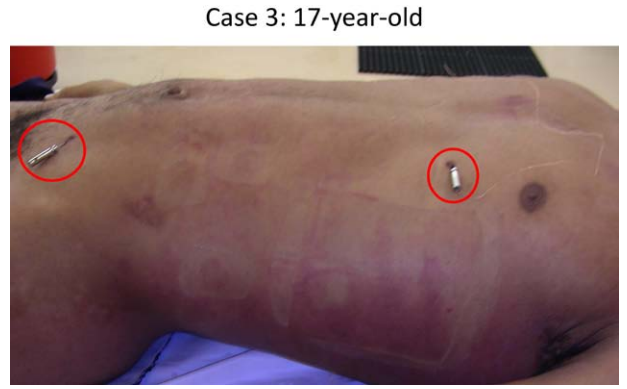

Figure 1. Picture at autopsy of case 3 with TASER X26 barbs still in place (circles). The heart at autopsy was normal.

After publication, 3 Letters to the Editor by physicians having TASER relationships disputed aspects of single cases but not the overall concept of TASER-induced VF. As I concluded in my response to those letters,[4] "…the published body of evidence now makes it perfectly clear that a TASER X26 ECD shock can induce VF in humans, transforming the argument from if it can happen to how often it happens."

Cases 7 and 8 from the original article[2] are expanded here to demonstrate causality and to make several points.

Case 7

A 16-year-old black boy (body mass index [BMI] 23 kg/m^2) with attention deficit disorder and asthma but without previous cardiac history or symptoms of heart disease ran ≈290 yd to an abandoned house. Confronted by police, possibly sweating, he received a 5-second chest shot with a TASER X26 from 3 to 4 ft away, immediately dropped to the ground, and was unconscious and unresponsive. One officer noted transient moaning and an apparent seizure ≈30 seconds after the shock and found no carotid pulsations or respirations. After 1 to 1½ minutes of cardiopulmonary resuscitation [CPR], the officer noted a carotid pulse and spontaneous respirations that

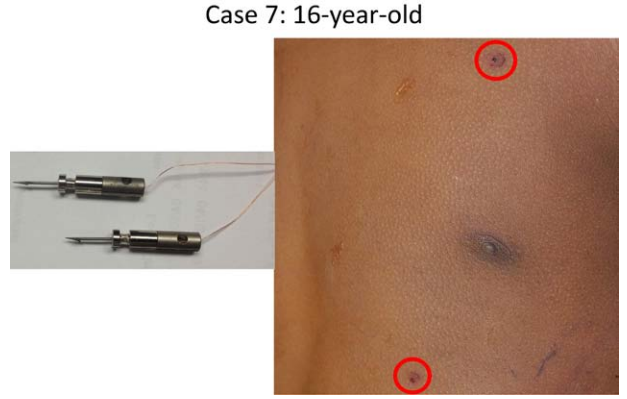

Figure 2. Left, Picture of 12.2- and 13.2-mm TASER X26 darts used in case 7 after removal from the skin. **Right,** Picture of TASER X26 dart marks (circles) above and below the left nipple of case 7. The head is at the top.

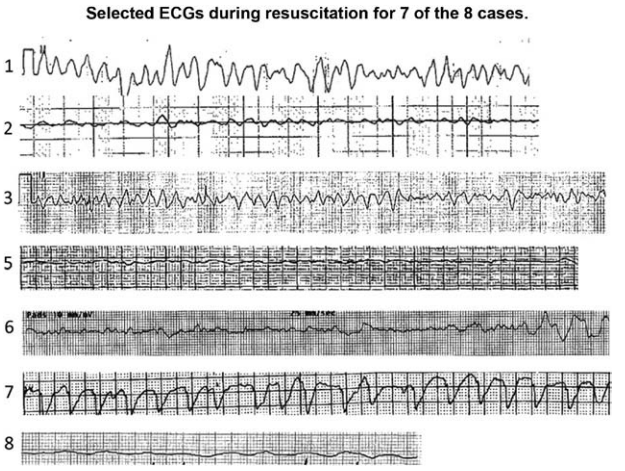

Figure 3. Selected ECGs recorded during resuscitation attempts in 7 of the 8 cases. Reproduced from Zipes. Sudden cardiac arrest and death following application of shocks from a TASER electronic control device. Circulation. 2012;125:2417–2422.[2]

lasted ≈15 seconds before ceasing spontaneously, and CPR was resumed. The first recorded ECG ≈8 minutes after the X26 shock showed VF. He was then defibrillated 4 times at 300 J and once or possibly twice at 360 J. He could not be resuscitated and was pronounced dead after transportation to the hospital. Autopsy showed 2 skin marks separated by 5¼ inches, consistent with TASER dart marks, 3 inches above and 2 inches below the left nipple (Figure 2 right). Dart length was 12.2 and 13.2 mm (Figure 2, left). The heart was slightly enlarged at 380 g, and the subject was diagnosed as having arrhythmogenic right ventricular cardiomyopathy (ARVC) by the forensic pathologist/medical examiner, who stated it was unknown whether the "taser [sic] device resulted in a direct effect on the heart or whether it served to exacerbate an

Figure 4. Case 8: still frames taken from video by police camera before and after TASER X26 administration. Initially, the man struggled with police (**left**). Off camera, he received X26 shocks of 21-, 7-, and 3-second duration and was then brought back into the video field by police (**right**). He was nonresponsive and gasping for breath, most likely agonal breathing. Note head at bottom of frame when he was unconscious, likely due to ventricular fibrillation. For full video, see Movie I in the online-only Data Supplement.

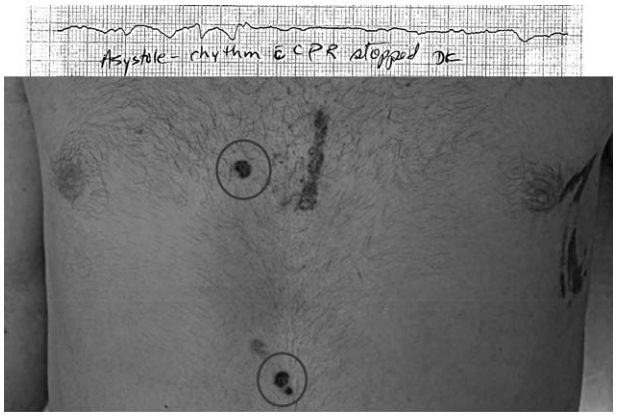

Figure 5. Top, ECG during resuscitation attempt. **Bottom**, Picture of case 8 at autopsy showing TASER X26 barb marks (circles).

arrhythmia….” A second pathologist agreed with the diagnosis of ARVC and stated that the “TASER ECD played no significant role in his death.” Another forensic pathologist detected “no convincing morphologic finding that can be construed as evidence of a preexisting abnormality of his heart” and indicated that the individual “...died as a result of an electric injury brought about by direct discharge of the Electronic Control Device….” Still another found that he did not have ARVC and that cause of death was a “cardiac dysrhythmia associated with the use of an electronic control device…a TASER.” Marijuana was the only drug found. He had been prescribed atomoxetine for attention deficit disorder, but its role, if any, is uncertain. Regardless of whether this young man had ARVC, he sustained a 5-second X26 chest shock with extra penetration darts near his heart (Figure 2), immediately lost consciousness, was noted to have VF ≈8 minutes later, and could not be resuscitated. If the alleged ARVC played a direct role, it would have had to trigger VF precisely when the X26 shock was delivered. In my opinion, it is more probable that the X26 shock caused the VF and subsequent death, and if the alleged ARVC was actually present, it facilitated VF induction.

Case 8

The police video (Figure 4 and Movie I in the online-only Data Supplement.) for this case helps establish causality. A 23-year-old white man (BMI, 25.5 kg/m^2) with no history of heart disease or cardiac symptoms struggled briefly with police officers after a traffic stop. He moved out of camera range for several minutes and received 3 X26 shocks of 21, 7, and 3 seconds. One police officer said the young man fell face forward after the first shock and was given a second shock because he could not or would not remove 1 of his hands from beneath his chest to be handcuffed. He was then carried back into camera range and propped curbside by police (Figure 4). Police officers said he initially was breathing and had a pulse. Despite CPR, an ECG recorded many minutes later documented asystole. At autopsy (Figure 5), cardiac scarring was noted (Table), but the cardiac pathologist could not state whether that contributed to his death. Blood alcohol level was 0.111 g/100 mL. The X26 deployment occurred between the left and right panels of Figure 4 and first and second parts of the video (see Movie I in the online-only Data Supplement). Scarring, if it played a direct role, would have had to trigger cardiac arrest precisely when the X26 shock was delivered. In my opinion, it is more probable that the X26 shock induced cardiac arrest and sudden death, and the scarring may have facilitated VF induction.

A More Recent Case

Since publication,[2] new cases have materialized, one of which is presented here because the police video again makes a compelling case of causality. A 50-year-old woman (BMI, 17.1 kg/m^2) with no manifest heart disease received a reported 3 X26 shocks to the left chest, with darts just above and below the left breast (based on dart marks noted subsequently). After the third X26 trigger pull reported by 1 officer, she immediately lost consciousness (video link of the X26 application can be found at http://www.youtube.com/watch?v=b6dHpt6c6zM) and started seizing. CPR was begun 6 minutes later, and an automated external defibrillator was applied 12 minutes after the X26 shock. After a shock for VF, she had return of sinus rhythm, breathed on her own, and survived, apparently with some memory deficit. Her drug screen was positive for cannabinoids but otherwise negative. Echocardiography shortly after resuscitation showed left ventricular end-diastolic dimension of 5.4 cm, estimated ejection fraction of 40%, mild to moderate mitral regurgitation with annular calcification, and mild to moderate tricuspid regurgitation consistent with myxomatous degeneration. Subsequent echocardiography showed normal LV size and systolic function, EF 55% to 60%, with thickened mitral and aortic valves, indicating most of the changes were likely related to the cardiac arrest. As in the 2 cases discussed above, this individual was fully active and conscious until receiving the X26 shock and then had a cardiac arrest caused by VF. If underlying heart disease played a direct role, it would have had to trigger VF precisely during the X26 shock. In my opinion, it is more probable that the X26 shock induced cardiac arrest and that any underlying cardiac pathology facilitated VF induction.

Incidence

The issue of how often cardiac arrest happens noted in my letter above is critical to establish the degree of risk. Amnesty International noted 334 deaths after an ECD shock between 2001 and 2008,[5] which increased recently to 544.[6] Although all in-custody deaths after ECD shocks are not likely a direct result of the shock, a number probably are. TASER International addressed that probability by revising their warnings from “aim at target: center of mass or legs” and “aiming at open front of unzipped jacket” before September 2009[7] to “when possible, avoiding chest shots…” after that date.[8] More recently, they noted that “heart rate, rhythm, capture” can occur and that “capture” and “cardiac arrest” can contribute to arrest-related death in physiologically or metabolically compromised persons.[9]

TASER counsel indicated that the risk of an ECD causing cardiac arrest was on the order of 1:100 000 applications.[10] Given an estimated 3 million TASER ECD applications,[10] this would compute to ≈30 deaths. However, the actual incidence of cardiac capture and cardiac arrest, and therefore the risk for this to occur, cannot be determined accurately for several reasons. First, as noted below, in the 2 instances of documented TASER ECD cardiac capture in humans, the individuals were totally asymptomatic during the 5- and 10-second exposure cycles. Thus, it is possible that transient cardiac capture occurs in the field but goes unnoticed if it does not result in cardiac arrest.

The second reason is the lack of accurate numbers to calculate incidence because no mandatory reporting exists in the United States. A bill requiring such reporting, for which I gave supporting testimony, was recently defeated by the Connecticut legislature (CGA H.B. No. 6628; 2013). The total number of TASER deployments is irrelevant because how often an individual is shot in the buttocks, abdomen, extremities, or back is of no cardiac concern. The number of chest shots is the important metric. One study[11] found that of 813 probe deployments, 21.9% had anterior chest placements. Obtaining an accurate estimate of incidence of death, and therefore risk from the TASER, would require an accurate estimate of the total number of deaths (numerator), a potentially underreported value, and the total number of chest shots (denominator), which is unknown. A recent article in the British newspaper *The Guardian* reported that of 884 TASER deployments from 18 of 45 UK forces since 2009, which was when TASER's warnings about avoiding chest shots were published,[8] 518 (59%) of all shots have hit the chest area.[12]

Animal Research

The most compelling evidence to prove the assertion made in the title of this article would be to record the development of VT/VF from a human during an X26 shock. This is very difficult for 2 reasons: The individual would require a cardiac recording device already in place during the shock, and the electric interference from the X26 could make any ECG recording unreadable. Therefore, animal studies become a necessary substitute.

A study in 2006[13] demonstrated that 5-second shocks from the equivalent of a standard TASER X26 ECD delivered via 9-mm darts inserted in various chest positions of anesthetized pigs caused cardiac capture, documented by an intracavitary right ventricular recording electrode. Dart vectors influenced capture. A position more likely to cause capture was from the sternal notch to the cardiac apex, resulting in ventricular capture ratios ranging from 6:1 to 3:1 (190–380 bpm). No VF occurred with normal output, but an increase in ECD power decreased the capture ratio, and VF consistently resulted when the ventricular capture ratio was ≤2:1. The authors noted that the data suggested

> …the potential for induction of ventricular tachycardia in subjects with substrate for ventricular tachycardia….Avoidance of this position would greatly reduce any concern for induction of ventricular arrhythmias.[13]

A second study using an off-the-shelf TASER X26 with a right ventricular recording lead showed that 52 of 53 discharges (98.11%) to the porcine chest caused cardiac capture, whereas 0 of 56 nonthoracic discharges stimulated the heart.[14] As with the prior study,[13] blood pressures fell to very low values at rapid capture rates. During epinephrine infusion to increase the spontaneous heart rate 50% to simulate the agitated stress state of an individual experiencing pain or resisting restraint, 13 of 16 TASER X26 discharges caused cardiac capture, 1 caused nonsustained ventricular tachycardia that spontaneously reverted to sinus rhythm, and 1 caused VT that evolved to VF and cardiac arrest (Figure 6).

In a series of 3 studies using 12-mm darts, investigators exposed pigs to two 40-second discharges from a TASER X26 ECD separated by a 10-second pause with ventilation between shocks. Five minutes after the shocks, pigs were profoundly acidotic with pH values of 6.86. One pig developed 3 minutes of sustained, monomorphic VT after the ECD discharge before finally progressing to VF (Figure 7).[15] After a left anterior thoracotomy to video the heart during the ECD shock, another pig developed VT proceeding to VF (http://www.youtube.com/watch?v=PxcXwk4UHm4). The second study[16] showed that succinylcholine eliminated the acidosis after the

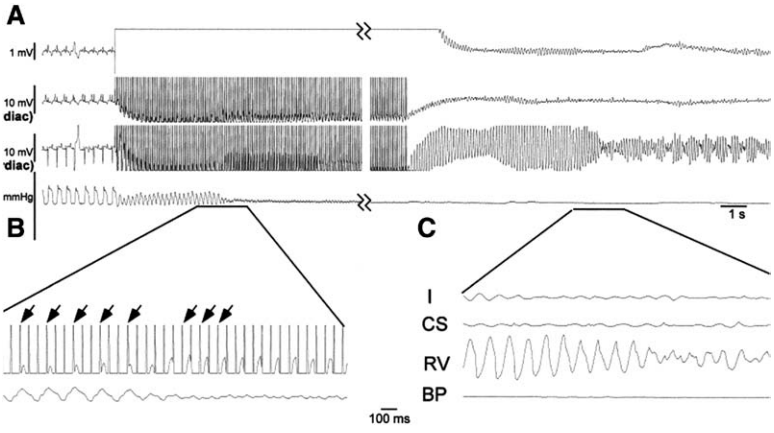

Figure 6. A, Recordings during X26-induced ventricular fibrillation (VF) in a pig while infusing epinephrine. **B**, Expanded time scale. The **arrowheads** at **left** depict a 3:1 response to the X26 discharge that progressed to a 2:1 response (**right**), which resulted in (**C**) rapid ventricular tachycardia (VT), degenerating into polymorphic VT and VF. Recordings are surface ECG lead 1, intracardiac electrograms from the coronary sinus (CS) and the right ventricular (RV) apex, and blood pressure (BP) from a Millar catheter in the descending aorta. Reproduced from Reference 14 with permission from the publisher. Nanthakumar K, Billingsley IM, Masse S, Dorian P, Cameron D, Chauhan VS, Downar ED, Sevaptsidis E. Cardiac electrophysiological consequences of neuromuscular incapacitating device discharges. *J Am Coll Cardiol.* 2006;48:798–804. Copyright © 2006, Elsevier.

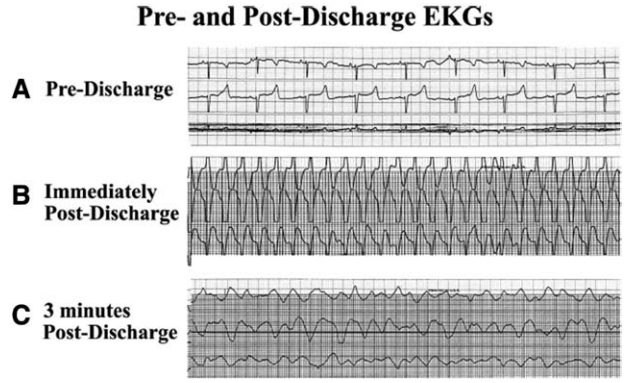

Figure 7. ECGs from a pig taken before (**A**) and after (**B** and **C**) TASER X26 discharge. An initial stable monomorphic ventricular tachycardia was induced after the X26 shock and remained for ≈3 minutes before evolving to ventricular fibrillation. Reproduced from Reference 15 with permission from the publisher. Dennis AJ, Valentino DJ, Walter RJ, Nagy KK, Winners J, Bokhari F, Wiley DE, Joseph KT, Roberts RR. Acute effects of Taser X26 discharges in a swine model. *J Trauma.* 2007;63:581–590. Copyright © 2007 Lippincott Williams & Wilkins, Inc.

X26 shock. One pig developed VF after a single 40-second ECD discharge. In the third study,[17] a 10-second TASER X26 discharge induced cardiac capture in 23 of 27 attempts over transcardiac vectors and induced VF in 2 of 4 animals. Nonsustained VT occurred after discharge in the remaining animals. The authors stated:

> If our data can be translated to humans, then ventricular rhythm may be captured and postdischarge dysrhythmias or VF may occur. Such transcardiac vectors should be avoided when possible and the potential for deterioration of the cardiac rhythm to VF in the field should be considered….Users should be trained to recognize the possible cardiac effects and be prepared to use automated external defibrillators and cardiopulmonary resuscitation maneuvers when needed.[17]

Recent reanalysis[18] of data from the earlier study[14] demonstrated that the average time for cardiac capture was 121 milliseconds (2 impulses from the stun gun), whereas 14 of 38 discharges (37%) captured the heart with the first impulse. The capture rate during discharge accelerated from 4:1 to 3:1 in 7 cases and from 3:1 to 2:1 in 2 cases in an average time of 3.6 seconds. Nonsustained VT followed 4 discharges. Both direct and indirect (via retrograde conduction from captured ventricular beats) atrial capture could occur,[19] and this caused an atrial arrhythmia on 1 occasion. These observations provide support for a 5-second shock being capable of inducing VF and the development of atrial fibrillation after a TASER shock.[20]

Drive-Stun Capture

Two porcine studies documented cardiac capture after X26 exposure in a drive-stun mode with probes taped to the skin between the suprasternal notch and point of maximal impulse[21] or 1 dart on the right chest and the other over the left upper abdomen.[17] In the latter study, darts held ½ in away from the skin by insulating foam blocks were still capable of producing cardiac capture. TASER International states that the high voltage used allows 1 probe to arc through a cumulative 2 in of clothing and not have to physically penetrate the body to have an effect (TASER Instructor Certification Course V-13, May 1, 2006, slide 35).

Thus, much like transthoracic cardiac pacing used emergently to treat bradyarrhythmias in humans,[22] pulses from the TASER X26 ECD emit sufficient electric charge to produce transthoracic cardiac capture despite high skin resistance. Because of this, drive-stun application should be capable of causing cardiac capture and VF in humans and was the focus of previous litigation (Williams versus TASER International, Inc; US District Court for the Northern District of Georgia; case No. 06-cv-00051 RWS; filed January 9, 2006). Darts that penetrate the high skin resistance should, in all likelihood, cause cardiac capture even more easily.

Mechanism of Cardiac Arrest

The porcine studies show that the mechanism by which the X26 provokes cardiac arrest is by capturing the heart and increasing its rate to values too rapid for maintenance of organized electric activity, resulting in VT/VF (Figures 6 and 7). Runaway pacemakers years ago produced the same phenomenon,[23] as does rapid pacing during electrophysiological studies. Thus, it should come as no surprise that transcutaneous rapid pacing from an X26 can accomplish the same thing. Ischemia from very low blood pressure could contribute to developing VF. A stimulated ventricular complex falling in the vulnerable period of the previous beat could theoretically induce VT/VF as well. Importantly, animal data (Figures 6 and 7) show that VT can precede the development of VF by seconds to minutes.[14,15] Therefore, after an X26 shock, an individual could have a palpable pulse for a variable time interval before lapsing into pulseless VT/VF.

Dart-to-Heart Distance

As with all cardiac stimulation, the distance between the stimulating electrodes and the myocardium is critical. When fired, the 2 TASER darts spread at an 8° angle, separating by ≈1 ft for every 7 ft of travel,[1] so the distance between 2 impaled darts can be fairly great. According to 1 study, 15 cm was the ideal spread distance for cardiac capture.[21] Although nonphysiological porcine studies have suggested that dart-to-heart distances of 4 to 17 mm are required to produce VF,[24,25] TASER ECD shocks with 1 dart in the right chest and the second in the abdomen or right groin, distances exceeding 4 to 17 mm, have been shown to capture the heart in intact pigs[17] and humans.[26] Rahko,[27] evaluating skin-to-heart distance by echocardiography, stated, "An EMD dart penetrating the skin directly over the heart might put individuals at risk for ventricular fibrillation" and noted that the skin-to-heart distance correlated with BMI. Using the porcine finding[24] of 26-mm skin-to-heart distance as a threshold value for individuals

potentially vulnerable to X26-induced VF, he found that 79% of nonobese individuals with BMIs <25 kg/m^2 were at risk.

In addition to anatomy, body position can influence skin-to-heart distance and therefore dart-to-heart distance. For example, changing positions from upright to prone can shorten the anterior chest-to-heart distance by almost 1 cm (H. Feigenbaum, MD, personal communication, 2013) and facilitate cardiac capture by darts in the anterior chest. Falling prone could drive the darts deeper into the skin. Lying on one's left side brings the heart so close to the chest wall that the apical impulse lies virtually just beneath the skin and is visibly seen and palpated. A dart over the apical impulse would be only a few millimeters from the heart. An enlarged heart could shorten skin-to-heart and therefore dart-to-heart distance. In addition, a state of excitation, for example, adrenaline released during an agitated state of fight or flight, can make the heart more susceptible to cardiac stimulation, which can facilitate capture or VF induction[14] (Figure 6). Because of these variables and the effect of the vector encompassed by the 2 darts,[13,14,17] in my opinion, no absolute number exists beyond which chest darts could not capture the heart in a particular individual. Darts closest to the cardiac silhouette would pose the greatest risk for cardiac capture and therefore VF induction.

Clinical Research

Multiple clinical studies of varying shock durations, placements, and measurements have not been reported to induce VF. However, because of ethical considerations to protect the volunteers from risk, none of these trials can replicate the actual clinical situation experienced by stressed individuals involuntarily receiving chest ECD shocks in the chaos of a field setting, especially if the shocks are repeated or lengthy. Moscati et al[28] tested supine individuals with 15-second TASER X26 shocks over "leads placed on the right upper chest and right upper abdomen" after alcohol ingestion and found a decrease in pH and bicarbonate and an increase in lactate after alcohol ingestion, with a further increase in lactate (mean, 4.19 mmol/L) and decrease in pH (mean, 7.31) after X26 exposure. No VF resulted. Dawes et al[29] studied volunteers with 15-second TASER X26 shocks without probe penetration by taping the conducting wires to the right upper chest and the right upper abdominal quadrant. Core body temperature did not change, and no VF resulted. One subject was excluded because of a history of coronary artery disease with 2 cardiac stents and frequent atrial and ventricular extrasystoles immediately before testing. Apparently, the authors recognized that this individual would be at risk for developing VF. Dawes et al[30] tested 5-second TASER X26 shocks delivered to 10 supine subjects (median BMI, 27.5 kg/m^2) over implanted chest darts (length not given) during echocardiographic monitoring. Heart rates before (mean, 91.0 bpm), during (mean, 95.8 bpm), and after (mean, 85.7 bpm) shocks did not show capture. The relatively slow mean heart rates are inconsistent with what probably happens during a law-enforcement confrontation in the field.

TASER ECDs can produce cardiac capture in humans. Cao et al[31] published a case report of a 53-year-old man with a dual-chamber pacemaker implanted subcutaneously beneath the left clavicle who received 2 X26 shocks with darts in the right chest. Pacemaker interrogation revealed 2 ventricular high-rate episodes that corresponded to the exact time of the X26 shocks. The man was asymptomatic. The ventricular electrograms during X26 cardiac capture were different from those during pacemaker capture, consistent with cardiac capture from the TASER shocks directly from the X26 and not over the pacemaker lead (L. Saxon, MD, personal communication, 2013).

The second study[26] tested a new-generation TASER ECD on normal supine human volunteers using echocardiographic monitoring. It demonstrated "an apparent brief episode of cardiac capture" at a rate of 240 bpm during the 10-second TASER ECD shock. The individual had no symptoms during the capture. One dart was slightly to the right of the midline chest with a skin-to-heart distance of 2.57 cm; the second was in the right groin.

Although neither individual developed VF, the fact that a TASER ECD could induce cardiac capture at rates exceeding 200 bpm makes it plausible that, in a given situation and given individual, perhaps in the presence of underlying heart disease such as an old myocardial infarction or a chemical substance such as alcohol or perhaps after longer or repeated shocks or during heightened sympathetic tone, TASER ECD–induced VF becomes possible, as previous authors have suggested.[13,17,27] Establishing that TASER X26 shocks cannot provoke VF would require replicating potential elements of field situations: testing shocks, some with >15-second duration, with 12- to 14-mm darts over a cardiac vector in multiple upright/prone volunteers, some with heart disease or after ingestion of potentially arrhythmogenic drugs or after receiving epinephrine. Such a study would require anesthetized patients in whom VF induction is necessary for ICD implantation testing.

The videos (Figure 4, Movie I in the online-only Data Supplement, and video at http://www.youtube.com/watch?v=b6dHpt6c6zM) in essence show the results of such clinical studies. Although there is no ECG recording or video of the heart, these videos of an "intact human" serve as a substitute for those experiments that cannot be done ethically and show what can happen in real life during a TASER X26 deployment.

Clinical Epidemiology

Several epidemiological studies have not concluded that TASER shocks induced VF. Gardner et al[32] reported that 100 shocks in subjects 13 to 17 years old caused no significant injuries. Eastman et al[33] found 1 death in 426 uses that may or may not have been causally related to the ECD. Strote et al[34] noted that, of 1101 individuals subjected to M26 and X26 shocks, none died. Bozeman et al[35] reported on 1201 uses, almost all X26s. Deaths of 2 subjects were not attributed to electric weapon use because of "prolonged combative behavior, cocaine use, cardiac abnormalities, and possible olanzapine toxicity," questionable reasons to exclude an ECD death. Apparently using the same cohort,[11] they noted "no immediate deaths in any cases…to suggest a cardiac dysrhythmia…."

These surveys are too small to exclude a TASER X26 risk of inducing VF. Even so, given the claim by Amnesty International[6] of 544 deaths associated with ECD use between 2001 and 2013, it is surprising that these studies did not capture at least some of these deaths, which brings their validity into question. Swerdlow et al,[36] using an Internet-based search, found 200 cases of ECD-associated, nontraumatic sudden deaths from 2001 to 2008. Thus, more deaths occurred after ECD deployment than captured by the epidemiological studies.

Prior Publications of Cases Consistent With TASER-Induced Cardiac Arrest

Kim and Franklin[37] reported that a 14-year-old adolescent shocked with a TASER X26 immediately collapsed and was found by paramedics to be in VF 2 minutes later. Four resuscitative shocks and drug administration restored a perfusing rhythm, and the adolescent made a nearly complete recovery. The ECG published, showing VF terminating after a 360-J defibrillation shock, was not the final shock, but 1 depicting an earlier 200-J shock converting VF to an idioventricular rhythm. Kroll et al[38] contested the accuracy of this report, but on the basis of both the paramedic's report and her deposition testimony,[39] the allegations of Kroll et al were shown to be incorrect. The paramedic testified that, immediately after the young man lost consciousness, she noted a pulse and respiration but recorded VF about 2 minutes later. A review by the National Institute of Justice[40] concluded for this case, "The proximity of collapse to CED (conducted energy device) use and documented VF argues in favor of an electrically induced cardiac event."

Another observation is of a 17-year-old boy who received TASER X26 shocks of 25 and 5 seconds in the anterior chest, immediately dropped to the ground, and became cyanotic and apneic.[41] The initial rhythm recorded >10 minutes later was asystole. Resuscitation included hypothermia, and he survived with memory impairment.

Of the 200 deaths analyzed by Swerdlow et al,[36] 56 subjects collapsed within 15 minutes of the ECD shock and had the presenting rhythm reported. Four had VF and 52 had bradycardia/asystole or pulseless electric activity. Swerdlow et al concluded that 1 death was typical of electrically induced VF and stated,

> For subject 1, who collapsed immediately…neither drugs nor cardiac disease can be implicated; both the time course and the electrode location are consistent with electrically induced VF." They continued, "To the best of our knowledge, this is the first reported fatality suggestive of [ECD]- induced VF.

Role of Underlying Heart Disease

Some of the TASER X26 ECD–induced cardiac arrests occurred in individuals alleged to have structurally abnormal hearts or in the presence of potentially arrhythmogenic substances such as alcohol.[2] Invasive electrophysiological testing over many years has demonstrated that it is easier to electrically induce VF when the heart is abnormal or in the presence of arrhythmogenic substances.[42] So, rather than preclude a diagnosis of X26-induced cardiac arrest in such a setting, the presence of these abnormalities actually helps support that diagnosis. Arguments suggesting that heart disease or a chemical substance, not the ECD shock, caused the cardiac arrest must require that coincidentally at the exact time of the TASER shock, the underlying heart disease or drugs triggered the VF, an unlikely assumption. Some individuals may have pacemakers[31] or defibrillators[43] in place, and they can be at risk for device-device interactions.

Importance of Vital Signs and Movement During VT/VF

Most observers, including physicians, rarely witness a person dying of VT/VF without intervening and do not know what to expect in terms of body movement, pulse, or respiration. In the unfortunate death of basketball player Hank Gathers resulting from exercise-induced VT captured on video, almost a full minute elapsed from the time he fell on court, presumably from a syncopal episode, until he finally stopped moving. He exhibited apparently purposeful movements, including sitting up, and breathing until finally succumbing to VF (www.youtube.com/watch?v=vcD5XUXfr1Y). Thus, claiming that a death cannot be due to VT/VF resulting from a TASER X26 shock because the individual was breathing or moving seconds or even minutes after the shock, when there can be no other cause for the sudden loss of consciousness in an individual who was alert and functioning immediately before the X26 shock, is without merit.

Multiple explanations exist for such events. First, VT before VF, as noted in several pig experiments[15] (Figures 6 and 7), could provide sufficient cerebral blood flow to maintain some bodily functions. In fact, in the case noted earlier,[37,39] the paramedic present during the entire TASER X26 deployment stated in sworn deposition testimony that she counted a pulse of 100 bpm over 15 seconds and respirations of 16 breaths per minute immediately after the TASER X26 ECD shock when the individual was totally unconscious, with VF established by ECG 2 minutes later (explained if VT with a pulse preceded the VF).

Second, normal breathing has been documented in sheep and pigs for as long as 1 minute and in humans for at least the first 12 to 15 seconds after the onset of VF.[44,45] Therefore, normal respirations can continue despite VF. Furthermore, confusing agonal respirations with normal respirations, especially early after VF onset, can confound the interpretation of the events.

Finally, accurate palpation of a pulse, particularly a radial pulse, in the midst of the turmoil of observing an unresponsive subject after a police altercation can be inaccurate. In a test among first responders checking a carotid pulse in patients before and while undergoing cardiopulmonary bypass, when no pulse was present, 10% (6 of 59) did not recognize an absent carotid pulse. In fact, only 1 in 59 emergency medical technicians and paramedics identified pulselessness correctly in 10 seconds, making the authors conclude that "…recognition of pulselessness by rescuers with basic CPR training is time-consuming and inaccurate."[46]

In a study using a computerized mannequin, 64 experienced healthcare providers checked the carotid pulse for 10 or 30 seconds. When there was no pulse, 27 of 42 responders checking for 10 seconds said there was a pulse, and 32 of 50 said there was a pulse after checking for 30 seconds. The authors stated,

> If the absence of a pulse was the only factor determining the onset of CPR maneuvers, approximately 50% of pulseless patients simulated in our study would not have had CPR initiated.[47]

Excited Delirium

Excited delirium has been reported as the cause of TASER X26–related deaths as a result of an agitated and irrational state, usually compounded by physical restraint. The diagnosis of excited delirium is not recognized by the American Medical Association as a medical or psychiatric condition but is recognized by the National Association of Medical Examiners. Many of the individuals dying with this alleged diagnosis have taken stimulant drugs such as phencyclidine, methamphetamine, and cocaine or have suffered from severe mental illness, were restrained with hands bound behind them and legs shackled, and held prone on the ground, making breathing difficult. Drug toxicity or postural hypoxia or anoxia has been appropriately suggested as contributing to death in many of these individuals. The presence of increased body temperature is said to be an important differentiator of excited delirium from other causes of death[48]; however, "the exact signs and symptoms [of excited delirium] are difficult to define precisely,…"[49] thus hampering an accurate diagnosis. It is possible that excited delirium is a form of takotsubo syndrome,[50,51] which might be a cause of some in-custody deaths. However, as noted above, to attribute an ECD death to excited delirium, one must postulate that the excited delirium, if the entity exists, caused VF at the precise time of the TASER shock.

Asystole

Untreated, VF evolves to asystole, sometimes in as short as 3 minutes[52] but usually longer,[53] and has been noted in some patients after ECD-related collapse[2,36] (Table and Figures 3 and 5). Waalewijn et al[54] analyzed 873 patients and found the probability to record VF decreased per minute and the probability of asystole increased as time from collapse elapsed. At 10 minutes, the probability of asystole without basic life support is ≈25%, rising to ≈35% at 15 minutes. Therefore, recording asystole after a prolonged "down time" following X26 administration does not exonerate the X26 from causing the death.

Causality

A temporal association alone does not prove causality. However, when the following exist, in my opinion, a causal relationship between the TASER X26 ECD and cardiac arrest in humans is established: (1) known causal mechanism (cardiac capture at rapid rates), (2) temporal association with loss of consciousness and subsequent cardiac arrest (TASER shock precedes both), (3) recorded VF (or asystole if a prolonged interval until first ECG; biological mechanism), (4) TASER shock(s) with 1 or both chest barbs near the heart (required for cardiac capture), (5) no other plausible alternative explanation (normal heart or underlying heart disease/drugs, if present, unlikely to cause VF at that precise time), and (6) similar cases in the literature (see above).

Conclusions and Recommendations

The animal and clinical data clearly support the conclusion that a TASER X26 shock can produce VF in humans by the mechanisms elaborated above. Although the risk may be low, its number cannot be known without universal record keeping and the creation of a national database. Because of this risk, it has been suggested that law-enforcement experts reassess ECD use to maintain a balance of safety for subjects and officers while still achieving the goal of maintaining law and order.[55] In this regard, the Cincinnati Police Department has revised its use-of-force policy to ban TASER chest shots except in self-defense or the defense of another.[56]

The use of TASERS may be increasing. A recent *Guardian* article indicated that the deployment of TASER weapons has more than doubled in England and Wales, from ≈3500 in 2009 to 14 500 in 2010 and 2011.[57] In addition, a new TASER ECD, the X2, capable of shooting 2 cartridges, has been tested in 4 pigs exposed to 5-second shocks; it produced cardiac capture in 17 of 71 exposures (24%) at heart rates of 206 to 313 bpm compared with X26 capture in 45 of 71 exposures (63%) at heart rates of 180 to 313 bpm.[58] No pig developed VF. The authors concluded that the "transcardiac" pathway was less important for capture than the proximity of the dart to the heart.

I think ECD manufacturers should undertake an educational campaign to make all ECD users aware of the VF risk. Educational material should stress avoiding chest shots if possible and should warn against repeated or long trigger pulls. However, it is clear that a single 5-second shock can induce VF. A user should be judicious with ECD deployment and treat it with the same level of respect as a firearm, suspect cardiac arrest in any individual who becomes unresponsive after a shock, quickly call for medical support, and be prepared to resuscitate, including using an automated external defibrillator if needed. A national database should be mandated.

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Disclosures

The Institutional Review Board of the Indiana University School of Medicine approved the medical information used for this article. Written informed consent was obtained from each person or an authorized representative. The cases included have been studied as part of litigation related to administration of ECD shocks from the TASER X26 device. I have served (and in the future may serve) as a paid

plaintiff expert witness in ECD-related sudden cardiac arrest/death cases. Despite this conflict of interest, I have tried to present the salient facts about the cases and to offer scientific evidence, credible argument, and logic to support the conclusions. Statements in this manuscript are my opinion, made to a reasonable degree of medical certainty.

References

1. TASER Electronic © 2009; TASER International, Inc. Control Devices Electrical Characteristics-X26. February 1, 2009.
2. Zipes DP. Sudden cardiac arrest and death following application of shocks from a TASER electronic control device [erratum appears in *Circulation*. 2012;125i:e27]. *Circulation*. 2012;125:2417–2422.
3. Myerburg RJ, Goodman KW, Ringe TB 3rd. Electronic control devices: science, law, and social responsibility. *Circulation*. 2012;125:2406–2408.
4. Zipes DP. Response to letters regarding article, "sudden cardiac arrest and death following application of shocks from a TASER electronic control device." *Circulation*. 2013;127:e261–e262.
5. *USA: "Less Than Lethal"? List of Deaths Following Use of Stun Weapons in US Law Enforcement: 3 June 2001 to 31 August 2008*. New York, NY: Amnesty International Publications; December 16, 2008. Index No. AMR 51/010/2008.
6. Electronic Village Blog. http://electronicvillage.blogspot.com/2009/05/taser-related-deaths-in-united-states.html. Accessed August 23, 2013.
7. TASER X26 electronic control device user course version. May 1, 2006.
8. TASER Training Bulletin 15.0. Medical research update and revised warnings. September 30, 2009.
9. TASER 2011 instructor and user warnings, risks, liability release, and covenant not to sue. May 31, 2011.
10. Brave M. Liability Assessment Awareness International, Inc. April 9, 2012.
11. Bozeman WP, Teacher E, Winslow JE. Transcardiac conducted electrical weapon (TASER) probe deployments: incidence and outcomes. *J Emerg Med*. 2012;43:970–975.
12. Malik S, Mole C. Police continued to fire Tasers at chests–despite cardiac arrest warnings. *The Guardian.* July 14, 2013. http://www.theguardian.com/uk-news/2013/jul/14/police-tasers-cardiac-arrest-warnings. Accessed August 23, 2013.
13. Lakkireddy D, Wallick D, Ryschon K, Chung MK, Butany J, Martin D, Saliba W, Kowalewski W, Natale A, Tchou PJ. Effects of cocaine intoxication on the threshold for stun gun induction of ventricular fibrillation. *J Am Coll Cardiol*. 2006;48:805–811.
14. Nanthakumar K, Billingsley IM, Masse S, Dorian P, Cameron D, Chauhan VS, Downar E, Sevaptsidis E. Cardiac electrophysiological consequences of neuromuscular incapacitating device discharges. *J Am Coll Cardiol*. 2006;48:798–804.
15. Dennis AJ, Valentino DJ, Walter RJ, Nagy KK, Winners J, Bokhari F, Wiley DE, Joseph KT, Roberts RR. Acute effects of TASER X26 discharges in a swine model. *J Trauma.* 2007;63:581–590.
16. Walter RJ, Dennis AJ, Valentino DJ, Margeta B, Nagy KK, Bokhari F, Wiley DE, Joseph KT, Roberts RR. TASER X26 discharges in swine produce potentially fatal ventricular arrhythmias. *Acad Emerg Med*. 2008;15:66–73.
17. Valentino DJ, Walter RJ, Dennis AJ, Margeta B, Starr F, Nagy KK, Bokhari F, Wiley DE, Joseph KT, Roberts RR. Taser X26 discharges in swine: ventricular rhythm capture is dependent on discharge vector. *J Trauma.* 2008;65:1478–1486.
18. Masse S, Desfosses-Masse J, Hado H, Waxman MB, Nanthakumar K. Determining the safe duration for stun gun discharge across the chest. *Heart Rhythm*. 2013;10:S186.
19. Hado HS, Masse S, Das M, Gizurarson S, Khan F, Roshn J, Waxman M, Nanthakumar K. The effect of stun gun discharges on the atrium. *Heart Rhythm* 2013; 10:S404.
20. Multerer S, Berkenbosch JW, Das B, Johnsrude C. Atrial fibrillation after taser exposure in a previously healthy adolescent. *Pediatr Emerg Care*. 2009;25:851–853.
21. Lakkireddy D, Wallick D, Verma A, Ryschon K, Kowalewski W, Wazni O, Butany J, Martin D, Tchou PJ. Cardiac effects of electrical stun guns: does position of barbs contact make a difference? *Pacing Clin Electrophysiol*. 2008;31:398–408.
22. Sherbino J, Verbeek PR, MacDonald RD, Sawadsky BV, McDonald AC, Morrison LJ. Prehospital transcutaneous cardiac pacing for symptomatic bradycardia or bradyasystolic cardiac arrest: a systematic review. *Resuscitation*. 2006;70:193–200.
23. Nasrallah A, Hall RJ, Garcia E, Kyger ER, Hallman GL, Cooley DA. Runaway pacemaker in seven patients: a persisting problem. *J Thorac Cardiovasc Surg*. 1975;69:365–368.
24. Wu JY, Sun H, O'Rourke AP, Huebner S, Rahko PS, Will JA, Webster JG. Taser dart-to-heart distance that causes ventricular fibrillation in pigs. *IEEE Trans Biomed Eng*. 2007;54:503–508.
25. Wu JY, Sun H, O'Rourke AP, Huebner SM, Rahko PS, Will JA, Webster JG. Taser blunt probe dart-to-heart distance causing ventricular fibrillation in pigs. *IEEE Trans Biomed Eng*. 2008;55:2768–2771.
26. Ho JD, Dawes DM, Reardon RF, Strote SR, Kunz SN, Nelson RS, Lundin EJ, Orozco BS, Miner JR. Human cardiovascular effects of a new generation conducted electrical weapon. *Forensic Sci Int*. 2011;204:50–57.
27. Rahko PS. Evaluation of the skin-to-heart distance in the standing adult by two-dimensional echocardiography. *J Am Soc Echocardiogr*. 2008;21:761–764.
28. Moscati R, Ho JD, Dawes DM, Miner JR. Physiologic effects of prolonged conducted electrical weapon discharge in ethanol-intoxicated adults. *Am J Emerg Med*. 2010;28:582–587.
29. Dawes DM, Ho JD, Johnson MA, Lundin E, Janchar TA, Miner JR. 15-Second conducted electrical weapon exposure does not cause core temperature elevation in non-environmentally stressed resting adults. *Forensic Sci Int*. 2008;176:253–257.
30. Dawes DM, Ho JD, Reardon RF, Miner JR. Echocardiographic evaluation of TASER X26 probe deployment into the chests of human volunteers. *Am J Emerg Med*. 2010;28:49–55.
31. Cao M, Shinbane JS, Gillberg JM, Saxon LA, Swerdlow CD. Taser-induced rapid ventricular myocardial capture demonstrated by pacemaker intracardiac electrograms. *J Cardiovasc Electrophysiol*. 2007;18:876–879.
32. Gardner AR, Hauda WE 2nd, Bozeman WP. Conducted electrical weapon (TASER) use against minors: a shocking analysis. *Pediatr Emerg Care*. 2012;28:873–877.
33. Eastman AL, Metzger JC, Pepe PE, Benitez FL, Decker J, Rinnert KJ, Field CA, Friese RS. Conductive electrical devices: a prospective, population-based study of the medical safety of law enforcement use. *J Trauma.* 2008;64:1567–1572.
34. Strote J, Walsh M, Angelidis M, Basta A, Hutson HR. Conducted electrical weapon use by law enforcement: an evaluation of safety and injury. *J Trauma.* 2010;68:1239–1246.
35. Bozeman WP, Hauda WE 2nd, Heck JJ, Graham DD Jr, Martin BP, Winslow JE. Safety and injury profile of conducted electrical weapons used by law enforcement officers against criminal suspects. *Ann Emerg Med*. 2009;53:480–489.
36. Swerdlow CD, Fishbein MC, Chaman L, Lakkireddy DR, Tchou P. Presenting rhythm in sudden deaths temporally proximate to discharge of TASER conducted electrical weapons. *Acad Emerg Med*. 2009;16:726–739.
37. Kim PJ, Franklin WH Ventricular fibrillation after stun-gun discharge: letter to the editor. *New Engl J Med*. 2005;353:958–959.
38. Kroll MW, Calkins H, Luceri RM. Letter to the editor. *J Am Coll Cardiol*. 2007;49:732.
39. Hutchison J. Deposition in Circuit Court of Cook County, 21 June, 2007. *Ortega-Piron v Lopez*. 05 L 1643.
40. Study of Deaths Following Electro Muscular Disruption: NIJ Report. Washington, DC: US Department of Justice, Office of Justice Programs; May 2011.
41. Schwarz ES, Barra M, Liao MM. Successful resuscitation of a patient in asystole after a TASER injury using a hypothermia protocol. *Am J Emerg Med*. 2009;27:515.e1–515.e2.
42. Greenspon AJ, Stang JM, Lewis RP, Schaal SF. Provocation of ventricular tachycardia after consumption of alcohol. *N Engl J Med*. 1979;301:1049–1050.
43. Haegeli LM, Sterns LD, Adam DC, Leather RA. Effect of a Taser shot to the chest of a patient with an implantable defibrillator. *Heart Rhythm*. 2006;3:339–341.
44. Zuercher M, Ewy GA, Hilwig RW, Sanders AB, Otto CW, Berg RA, Kern KB. Continued breathing followed by gasping or apnea in a swine model of ventricular fibrillation cardiac arrest. *BMC Cardiovasc Disord*. 2010;10:36.
45. Haouzi P, Ahmadpour N, Bell HJ, Artman S, Banchs J, Samii S, Gonzalez M, Gleeson K. Breathing patterns during cardiac arrest. *J Appl Physiol (1985)*. 2010;109:405–411.

46. Eberle B, Dick WF, Schneider T, Wisser G, Doetsch S, Tzanova I. Checking the carotid pulse check: diagnostic accuracy of first responders in patients with and without a pulse. *Resuscitation*. 1996;33:107–116.
47. Lapostolle F, Le Toumelin P, Agostinucci JM, Catineau J, Adnet F. Basic cardiac life support providers checking the carotid pulse: performance, degree of conviction, and influencing factors. *Acad Emerg Med*. 2004;11:878–880.
48. Jauchem JR. Pathophysiologic changes due to TASER® devices versus excited delirium: potential relevance to deaths-in-custody? *J Forensic Leg Med*. 2011;18:145–153.
49. Vilke GM, Bozeman WP, Dawes DM, Demers G, Wilson MP. Excited delirium syndrome (ExDS): treatment options and considerations. *J Forensic Leg Med*. 2012;19:117–121.
50. Wittstein IS, Thiemann DR, Lima JA, Baughman KL, Schulman SP, Gerstenblith G, Wu KC, Rade JJ, Bivalacqua TJ, Champion HC. Neurohumoral features of myocardial stunning due to sudden emotional stress. *N Engl J Med*. 2005;352:539–548.
51. Madias C, Fitzgibbons TP, Alsheikh-Ali AA, Bouchard JL, Kalsmith B, Garlitski AC, Tighe DA, Estes NA 3rd, Aurigemma GP, Link MS. Acquired long QT syndrome from stress cardiomyopathy is associated with ventricular arrhythmias and torsades de pointes. *Heart Rhythm*. 2011;8:555–561.
52. Dubrey SW, Grocott-Mason R. Spontaneous recovery from prolonged cardiac arrest. *Heart*. 2002; 87:432.
53. Hallstrom AP, Eisenberg MS, Bergner L. The persistence of ventricular fibrillation and its implication for evaluating EMS. *Emerg Health Serv Q*. 1982;1:41–49.
54. Waalewijn RA, Nijpels MA, Tijssen JG, Koster RW. Prevention of deterioration of ventricular fibrillation by basic life support during out-of-hospital cardiac arrest. *Resuscitation*. 2002;54:31–36.
55. Miller ME. Taser use and the use-of-force continuum: examining the effect of policy change. *The Police Chief*. 2010; 77: 72–76.
56. O'Keefe PJ. Cincinnati police revises use of force policy, specifically with use of Tasers. September 18, 2012. http://www.wcpo.com/news/local-news/cincinnati-police-revises-use-of-force-policy-specifically-with-use-of-tasers. Accessed August 23, 2013.
57. Laville S. *The Guardian.* September 8, 2013. http://www.theguardian.com/uk-news/2013/sep/08/police-use-tasers-doubles-year. Accessed September 8, 2013.
58. Dawes DM, Ho JD, Moore JC, Miner JR. An evaluation of two conducted electrical weapons and two probe designs using a swine comparative cardiac safety model. *Forensic Sci Med Pathol*. 2013;9:333–342.

Response to Zipes

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In controversies like this, transparency, absence of conflict, and adherence to the scientific method are important requisites. In Dr Zipes' series, not a single medical examiner postulated the ECD as the primary cause of death. The presumably unbiased Council of Canadian Academies report (http://www.cahs-acss.ca/the-health-effects-of-conducted-energy-weapons-2/) concluded that Dr Zipes' "study…is particularly questionable" and stated that "In the [>2 000 000 ECD uses in the] field there has not been a conclusive case of fatal ventricular fibrillation caused solely by the electrical effects….A small number of human cases have found a temporal relationship between [ECDs] and fatal cardiac arrhythmias but they do not allow for confirmation or exclusion of a clear causal link…." Dr Zipes cites Sherbino to support his claim that the ECD charge is equivalent to that of transcutaneous pacing; however, Sherbino does not report any pacing thresholds. Zipes himself, with Klein as first author, reports thresholds of 2440 microcoulombs (61 mA·40 ms)—significantly greater than the 100 microcoulombs of the ECD. The temporal coincidence argument is discussed in our article and online-only Data Supplement and is refuted by Dr Zipes' case 4 (our case 8) in which the probes missed the subject. Dr Zipes may also be confusing the sensitivity of a pulse finding (which is low) with its specificity (which is high). The assertion of a "precise" timing (between the ECD and cardiac arrest) may be ill advised when the majority of cases had a documented pulse, normal breathing for 6.1±3.1 minutes, and abnormal underlying cardiac morphology.

TASER Electronic Control Devices Can Cause Cardiac Arrest in Humans

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Movie I

Case 8: Video taken by police camera before and after TASER X26 administration. Initially, the man struggled with police (first segment). Off camera, he received X26 shocks of 21-, 7-, and 3-second duration and was then brought back into the video field by police (second segment). He was nonresponsive and gasping for breath, most likely agonal breathing due to VF.